VOLUME LXVI APRIL, 1910 No. 4

AMERICAN JOURNAL OF INSANITY

THE NEW GOVERNMENT HOSPITAL FOR THE INSANE.

BY WILLIAM A. WHITE, M. D., SUPERINTENDENT, WASHINGTON, D. C.

Next May the American Medico-Psychological Association will meet in Washington. The Thursday afternoon session of the meeting will be held at the Government Hospital for the Insane and many of the members will, I trust, visit the hospital, so I am taking this opportunity of "writing up" a short account of the hospital, describing its principal features, particularly the medical and scientific organization, and setting forth those points that I think may be of interest to the members of the Association.

The hospital was created in 1855 by Act of Congress and since that time has been steadily growing. As might be supposed practically every type of asylum construction that has been in vogue for the past half century is represented in the several departments of the institution. While many of these features have a great deal of interest it is not the purpose of this article to dilate upon them but only to take up the more recent improvements in both construction and organization.

The new hospital extension, designed by Dr. Richardson, which has in large part made possible the improvements of recent years, comprises fifteen buildings which were under construction when I took charge October 1, 1903. These fifteen buildings include an administration building, a nurses' home for women, a detached kitchen, a power, heat and lighting plant, and eleven buildings for the accommodation of one thousand patients. The cost of this extension was approximately $1,500,000.

The first of these fifteen buildings was occupied in June, 1904. From that date the other cottages have been successively opened and this shifting of the patient population made it possible to remodel many of the older structures. Four buildings have been entirely remodeled; one made into a nurses' home for men, the

porches of several of the old buildings have been made into sun parlors (Fig. 1) and wards for the tubercular, a dispensary and circulating library established in the old administration quarters, two buildings abandoned, many small structures torn down, two buildings moved in their entirety to different locations, and an amusement hall with seating capacity of approximately 1200 erected. At the present time the three power plants of the hospital are being consolidated and the electrical system changed from the direct to the alternating current.

The eleven new cottages for patients are of two general types—small cottages for from 30 to 60 patients, and large cottages of four wards each for 120 patients. There are five cottages of this latter type, one each for the disturbed classes, male and female, one for the infirm and bed-ridden white men, and two psychopathic (receiving) buildings. These two psychopathic pavilions are located one on either side of the new administration building (Fig. 2) and have certain features of construction that are worth noting (Figs. 3, 4, 5, 6). These features are shown in the plans and are: the porches, enclosed on the second floor, large enough to accommodate the entire population of the wards—a special corridor for noisy patients which can be shut off completely from the rest of the ward and has a continuous bath at one end—a surgical department consisting of surgical amphitheater, anesthetizing room, sterilizing room, and surgeons' room—a hydrotherapeutic equipment—and a large proportion of single rooms.

For administrative purposes the hospital is divided into five departments, each with a chief, as follows: Steward, Disbursing Officer, Chief Clerk, Clinical Director, and Scientific Director. The first three of these chiefs of departments have to do entirely with the administrative and business management of the institution and I will, therefore, not mention them further in this article.

The medical service of the hospital is presided over by the clinical director, a position which was created something over three years ago. His duties comprise the general supervision and oversight of the medical work of the hospital, and the charge of the hydrotherapeutic department, operating room, training school for nurses, and the clinical records. In general, his function is to bring into closer organic connection the different medical services and to that end he operates as a clearing house through which all transfers of patients from one service to another must be made.

As for the rest, the organization of the several services of the hospital is practically the same as in any other institution of this sort. There is, however, a large consulting staff of the best physicians in the city of Washington in the various departments of medicine. These gentlemen are called upon frequently by the hospital, especially the surgeons.

The scientific director presides over the scientific research work of the institution. The personnel of this department is as follows: A psychologist, pathologist, histopathologist, clinical pathologist, an assistant to the psychologist, and a technician.

The function of the scientific director are in the main to have administrative control of the laboratories and to direct the work therein with a view, so far as possible, of coordinating the efforts of the several investigators along similar lines of research.

The pathological laboratory building contains the mortuary, an anatomical amphitheater (Fig. 7), a museum, a photographic department, and laboratories for histopathology, clinical pathology, bacteriology, physiological chemistry, and a library and private laboratory for the pathologist.

In addition to the laboratories in the laboratory building, a psychological laboratory has been installed on the first floor of the Allison Building. This laboratory contains a library and private laboratory for the scientific director, a general laboratory room, and small rooms for special research work.

A special feature of the medical and scientific work is the medical library (Fig. 8), located in the administration building. While the library itself is small it contains a full set of the index of the Surgeon-General's library, and a duplicate card in its index of every medical book in the Library of Congress, which, with the Index Medicus, gives us a complete catalogue of practically all of the medical literature of the world. With all this literature, too, being right here in Washington it is available within two hours. In addition to the above the hospital subscribes for about forty of the leading journals which deal with psychiatry and related subjects.

Another special feature of the medical and scientific work is that six members of the medical and scientific staffs occupy teaching positions in the medical colleges of the city. This relationship with the medical colleges has always been encouraged, and it is believed to be an excellent thing for the hospital.

The unique position in which the Government Hospital stands in the country is such that its possibilities for usefulness are very great. An attempt has been made to develop these possibilities, particularly with reference to the federal departments. A few years ago an arrangement was made for interchange between the medical officers of the hospital and the Public Health and Marine Hospital Service for the purpose of securing men trained in psychiatry to serve on Ellis Island in the detection of insane immigrants. For approximately two years now the hospital has had a man stationed on Ellis Island for that purpose.

For some years past I have been convinced of the importance of the problem of insanity in the military organization of the country and have finally succeeded in securing recognition of its importance in a material way. The surgeon-general of the army has detailed a medical officer to serve at the hospital, and his researches on the relations of the problem of insanity to the army have demonstrated fully the wisdom of this assignment. The Secretary of the Navy has recently also caused an assignment of a naval surgeon to the hospital.

In order that some idea of the work that is being carried on in the scientific department may be had, I will give a short sketch of the various investigations now being prosecuted in the several laboratories as follows:

Psychological Laboratory:

1. Psychogenic factors in certain mental disturbances.
2. Touch sensations in different bodily segments of normal people.
3. Sensation changes following local anesthesia.
4. Skin sensations following nerve division.
5. Comparative study of the reactions of different classes of the insane to stimuli of a painful character.
6. Sensory changes from trauma of the post-central cerebral convolutions.
7. The relation of visual sensations and perceptions to the occipital lobes.
8. Relation of the anterior and posterior cerebral association areas to the formation and retention of associations.
9. The character and time of associations in normal people.

10. The relation of sensory and motor defects to the education of certain feeble-minded.
11. The changes in volume and blood pressure of the cerebrum coincident with mental states.

Pathological Laboratory:

1. Study of the localized atrophies and scleroses of the brain in epilepsy.
2. Study of the idiopathic atrophies of the brain in various mental conditions, apparently not dependent upon arteriosclerosis, nor other vascular conditions.
3. A study of the various anatomical types of general paralysis, with special reference to the presence or absence of adhesions and decortications and hyperæmic and anæmic conditions.
4. Continued studies of the renal and adrenal growths, the hypernephric tumors especially.
5. Continued studies in the gross morbid anatomy of the epilepsies, and in pachymeningitis interna.

Histopathological Laboratory:

1. A study of the ventricular connective-tissue plaques.
2. The relationship between neuroglia cells and ganglion cells in the cerebral cortex. (Adaptation of the satellite cells with formation of Stäbchenzellen, Abräumzellen, and neuroglia baskets about the ganglion cells.)
3. Comparative study of the histological changes taking place in the Ammon's horn in epilepsy, paresis, senile processes and experimental conditions including hydrophobia.
4. Experimental study of the conditions leading to metachromatic changes of protoplasm.
5. Study of material of pellagra cases.

Clinico-Pathological Laboratory:

At the time of writing the clinical pathologist is in Vienna where, in collaboration with one of the University professors, he is prosecuting certain investigations into the changes brought about by syphilis in the central nervous system. These investigations are along the following lines: The syphilitic anaphylactic reaction, the cultivation of the treponema pallidum, and the globulin content of the blood.

Beginning with the first of the year a course of instruction was outlined to be given by the Scientific Department for the benefit

of the junior members of the medical staff. It has been so difficult to keep experienced men upon the medical staff, because of the great demand for such men throughout the country, that the hospital was confronted at the beginning of the year with a rather serious condition because most of the work had to be done by men only recently added to the staff. It was felt that a special effort ought to be made to bring these men to a condition of usefulness in a minimum length of time. To this end a series of lectures and demonstrations was outlined covering the fields of anatomy and gross pathology of the brain, histology and histopathology, clinical pathology, and neurologic and psychiatric methods. The course was eminently successful and fully warranted the efforts expended on it. An examination was held at the end of the course, which examination constitutes a part of the record of the different physicians and is a basis on which promotions are made.

In connection with the medical and scientific staffs, the hospital last winter started a series of evening staff meetings inviting the members of the medical profession of the city to the hospital and providing the entire program. The program was selected with a view to the needs of the general practitioner. Cases were presented such as might be met in every-day practice, and such recent aids to diagnosis were demonstrated as the Wasserman reaction and the Noguchi test for globulin in the cerebro-spinal fluid. As the result of these meetings a bulletin was published by the hospital containing the papers presented, which were distributed to the various institutions throughout the country in which mental disorders are treated. It is contemplated continuing this method of procedure during the current year, and in addition, endeavoring to enlighten the public by lectures as to the character of work the hospital is trying to do. One such lecture has already been given under the auspices of the local medical society.

I am constantly made aware of the suspicion in which hospitals for the insane are held and the consequent injustice which is done to the sufferers from mental disorder. Only very slowly is this suspicion being dispersed. To my mind one of the greatest works a hospital for the insane can do is to dispel these erroneous views in the community in which it is located, and I fully believe it is the duty of every hospital superintendent to exert himself to this end.

I trust this very brief summary of points of interest will prove interesting, and of service to our visitors in May.

FIG. 1.—Porch sun parlor.

FIG. 2.—Administration Building. Connecting corridors on either side lead to the psychopathic pavilions.

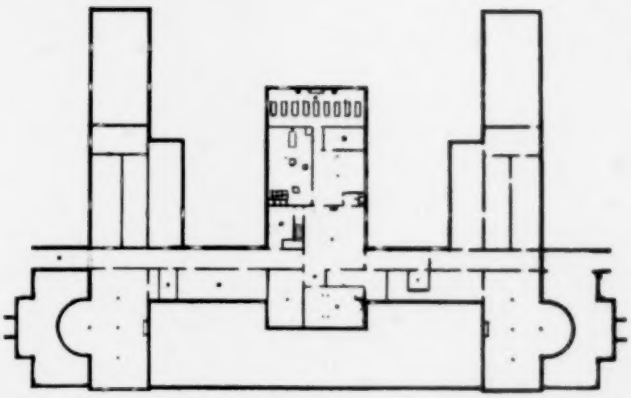


FIG. 3.—Psychopathic Pavilion. Basement plan. Shows location of hydrotherapeutic department in center.

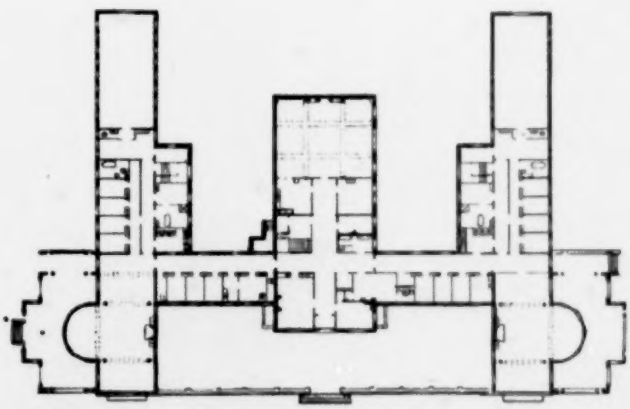


FIG. 4.—Psychopathic Pavilion. First-floor plan. Shows large proportion of single rooms, large porches, four rooms on left side in a special corridor for noisy patients with continuous bath located at end.

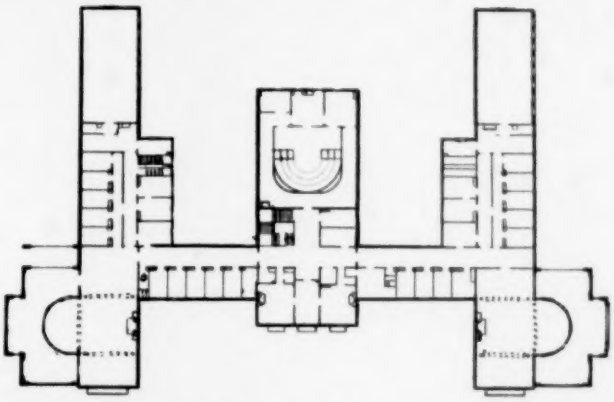


FIG. 5.—Psychopathic Pavilion. Second-floor plan. Shows location of operating amphitheater, etc., in center.

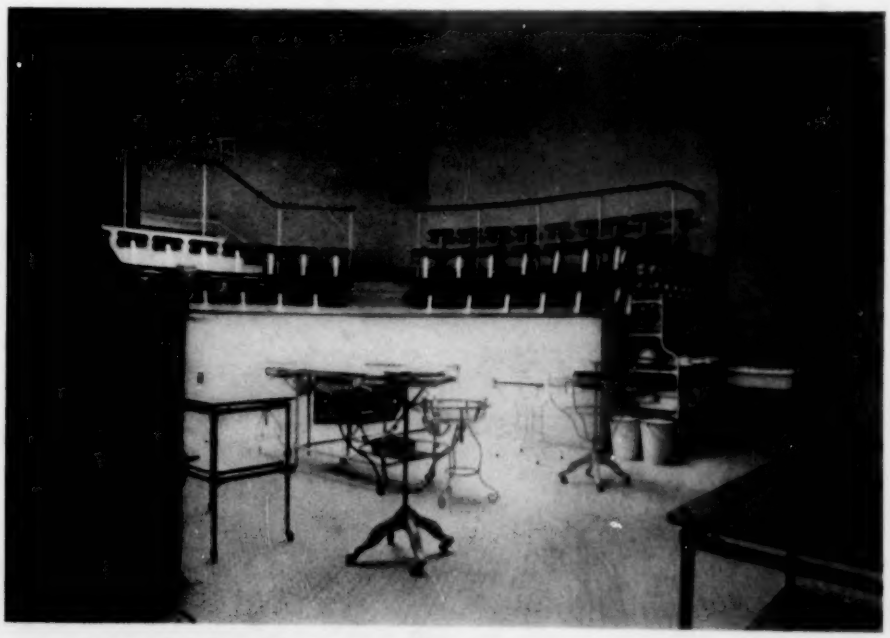


FIG. 6.—Surgical Amphitheater. Psychopathic pavilion for women.

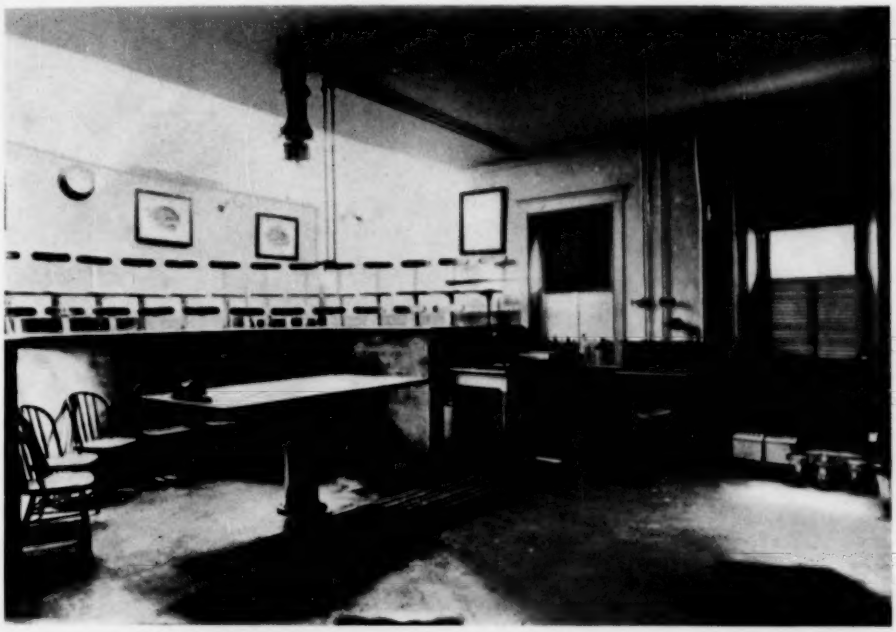


FIG. 7.—Anatomical Amphitheater.

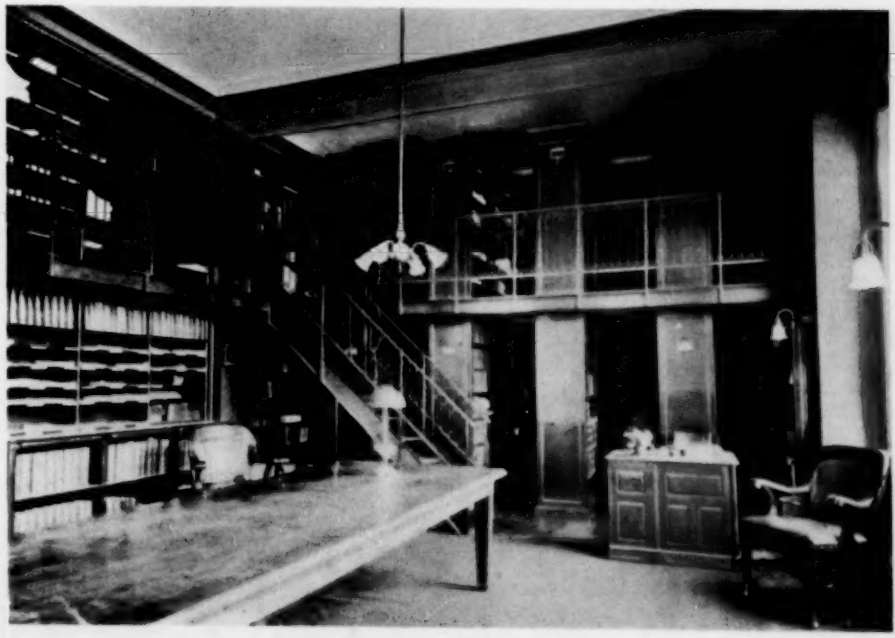


FIG. 8.—Medical Library.

A STUDY OF THE AUTO AND SOMATOPSYCHIC REACTION IN FOUR CASES OF DEMENTIA PRÆCOX.*

By WM. BURGESS CORNELL,

Assistant Physician, Sheppard and Enoch Pratt Hospital, Towson, Md.

The dementia præcox group seems ever widening to include increasing numbers of variations from the three older sub-types, hebephrenic, catatonic and paranoid. The clinical method of Kraepelin has been of an undoubted value in the comprehensive consideration of symptoms and description of the case as a whole, as well as in the grouping together of these cases to form a nosological entity upon which in a practical manner prognostic, diagnostic and therapeutic measures can be based.

The application of Wernicke's psychological analysis has lent further assistance in the description and study of single symptoms. Wernicke distinguishes primary and secondary identification or sensation, and assumes that secondary identification, which comprehends all the functions implied in mental grasp and elaboration, is the seat of disorder in all mental disease. Secondary sensation is divided into auto-, allo- and somatopsychic fields, or subjective consciousness as relates to the ego, its projection and environment, and its reciprocal relation to the body. Furthermore, disorders such as occur chiefly in the sensory sphere are termed psychosensory, those of thinking, intrapsychic and those mostly involving the motor mechanisms, psychomotor.

The elaboration of such methods has led to an individual psychology, which makes each case of mental disorder a variant from others, and through which Breuer and Freud, and later Bleuler and Jung, have inaugurated a new epoch in psychiatry by such works as the *"Diagnostische Association Studien," "Affectivität, Suggestibilität, Paranoia," "Psychopathologie des Alltagsleben"* and *"Die Traumdeutung."*

Tiling as well as Freud came early to the conclusion that the importance of the individuality in the origin and the formation of

*Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

the psychosis was almost boundless. Proceeding along these lines, Jung, in studying the association in dementia præcox, has come upon important and far-reaching facts and has even attempted to explain the etiology on psychological grounds. He has found an identity in the psychological mechanism of hysteria and dementia præcox. Both show an association complex which the individual tends to elaborate in every direction until he is forced out of all adaptation to his environment. In hysteria Jung is sure of the causal connection between the complex and the disease, but not so in dementia præcox. In the latter the complexes become more tenaciously fixed and he further postulates that the complex produces, in addition to its psychological effects, an "X" or metabolic toxin which completes the psychic mutilation. Yet Jung does not deny the possibility that the "X" may primarily result from other than psychological reasons.

As for the main facts in the psychopathology of dementia præcox, various observers preceding Freud have all called attention to the existence of a central disturbance, although different terminology has been used, such as "apperceptive dementia" (Weygandt), dissociation and *abaissement du niveau mental* (Janet), tendencies toward fixation (Masselon, Neisser) and affect dementia (Kraepelin).

Then came the demonstration by Freud of the presence of split-off series of ideas, their repression and subsequent reappearance and fixation as complexes.

Interesting and far-reaching as are the theories of Bleuler, Freud and Jung, it is apparent that the psychoanalytic method apparently does not disclose a complex in every case of dementia præcox to which the dissociation may be attributed. Especially is this true in the hebephrenic and catatonic forms. It is also apparently true in the four cases whose abstracts follow.

In considering etiology in such types as these one is more inclined to consider the "X" or toxin is of primary importance, rather than secondary to a psychological complex. The nature of such a toxin and the method of its action remain to be disclosed by a better chemistry and histopathology of the future.

CASE I.—Male, 19, single. Family history is bad on both sides. Both parents are intelligent and highly educated. Father was alcoholic at the

time of patient's conception, and has remained so since; at the time of the patient's admission to the hospital he was going through an attack of delirium tremens. Mother had some sort of psychosis at 18, lasting six months.

Patient is the sixth of seven children; the oldest is a talented musician, but erratic and eccentric; the third has probably an aborted form of dementia præcox; the others are said to be well. Our patient, up to the age of 14, was considered to be the most healthy and brilliant of all the children. In most of his school work he had difficulties, but at a precocious age he read Darwin, Spencer and Huxley. He was also musically talented, and at one time gave great promise as a violinist. He has always been of an argumentative turn of mind, and delighted to enter into weighty discussions on philosophical and metaphysical subjects; his sexual life was normal, as far as could be ascertained. Personal history in other regard is negative.

About 14 he contracted a fever, which may have been typhoid, though not identified as such. During convalescence he refused to eat and was very thin; he lived mostly on milk. He improved somewhat, but later, on reading some books on physical culture, he decided he would be stronger if he lived on a very limited diet. He was in a very nervous condition. After a few months' schooling his health demanded his withdrawal and he was sent to Florida, where he improved and was able to enter school the following fall. Soon he was again compelled to leave. His condition steadily grew worse, and at 17 he was sent to a sanitarium. Later he improved and again attempted school. He grew sluggish and indifferent mentally and was once more taken out.

In the spring of 1908, at the age of 19, he was sent to northern Maine for the summer. He then complained of a loss of feeling for things in general and of a general sense of unreality. He expressed himself volubly and intensely. He complained of the uncongeniality of his home surroundings. On his return from Maine the feeling of unreality was more marked; he complained of a general sense of vagueness, said at times he could not distinguish between his right and left hand. All mental processes seemed slow. There was a tremor of the lips, some cutaneous cyanosis, tongue coated and breath foul. Mental condition showed variability. He would suddenly change from an apparently bright and normal state to one in which he became confused and retarded, and at times impulsive.

Shortly after admission the following interrogatory was taken with the aid of a stenographer:

Dr. How do you feel to-day? Pt. I feel, I don't feel. Dr. Why don't you feel? Pt. (pause). Well, I don't know enough to tell you why I don't feel—I don't—it would take a man who knows a great deal, a very great deal about physiology, psychology and everything else to tell you the exact reason why I don't feel (pause). Don't you think it would (smiling)? Now, if you want me to talk I will talk all you want

(smiling). Dr. You told me last night you thought you were going to sleep. Pt. O, yes, I am, I gradually went to sleep last summer (pause), gradually went to sleep—deadened—poisoned—poison forming from malassimilation, and the poison is the result, not the cause—that is, the cause of the cause is primarily the deadening of all these nerves through here and the inability of these nerves to tell the brain that there is a pain in here (pointing to stomach), and that's as near as I can tell you. That is a month ago, six months ago, in the spring, when I would have—my food wouldn't really digest properly—there would be no pain and, of course, gradually grew worse—deadened. Osler could tell you what is the matter with me. What's this for, to see whether I am sane or insane (looking at stenographer and smiling)? Dr. No, I want to get a description of what you think. Pt. I could have told you perfectly two weeks ago, but since then I have deadened very much, all associations and memory, association and memory and (pause) various other things that go to make up what you choose to call self have gone; the idea of possession, that my arms and legs belong to me, that's gone—I don't feel anything at all—I appear to other people, I am not self at all. Dr. You feel that you have changed? Pt. No, I am nothing now, I have lost myself, I cannot go back, I cannot look back—there is no memory, really. Dr. Then you have changed? Pt. Of course, I have changed. Dr. How do you mean? Pt. I gradually stopped—I was 18 then, and weighed 80 pounds. I went on a milk diet and went up from 80 to 145 pounds—gained 50 pounds in two months—9 pounds in one week—most I ever gained; and while I was gaining that I gradually deadened, that is, there was a loss—I got fat, but the legs would get all swollen up, with water, I think. My brother and I went to school and I couldn't do any real studying; in other words, I got fat, but I didn't get strong. There is no real, there is no real motor energy about my—whatever you choose to call it—no nervous energy. Then in the spring, after going to school, and I was very tired, I had been tired all that winter—all the winter before—slept all the time, wanted to sleep and I (pause) gradually went more and more to sleep, and felt as if my head was made of rotten blood (pause). I have lost all memory and associations and ability to go back, and inner feeling, lost all that entirely, and all retention of anything, for instance, when I go to, go up (pause), go on a fishing trip to the lake and come back, I couldn't retain it. I don't retain scenes, ideas of position. Dr. You remember them though? Pt. Yes, I can remember, all that's good, but suppose you have a lot of intellect, but haven't any feeling, what then? Well I gradually deadened in the summer and a (pause) kept on getting as much sleep as possible, and when I would go to sleep my whole arm, this arm (showing physician) would go to sleep; I would have to take this arm and hit it and wake it up, but there is no real memory. Dr. You have periods in which you feel real stupid? Pt. Stupid! I am stupid all the time. Dr. You are not this afternoon. Pt. I am not? Well, what is it? Suppose you are the most intellectual

person in the world, but you can't get out and fight for your living, well, what's that? I wouldn't want to sit in a chair—I have had about a million different selves. Dr. Last night when you came in you talked very well and then gradually grew confused. Pt. I don't remember anything—I am outside of myself—I am in myself. I feel inside—rather automatical—there is no sense of contrast of being tired or being refreshed, or the hundred of other contrasts that go to make up life—that's all it is—contrast—eating and sleeping and being tired and being rested—having pleasure and having pain, and the keener the contrast the keener do you live. I have lost all relationship with people—they are not human. You see, it's that association and everything have gone, and what have you got? Suppose you go into a restaurant and eat and you have no ears or eyes, or anything, and are utterly out of relationship with everything. I cannot talk to you now rationally (pause). Dr. Did you have an unhappy home life? Pt. I have never known what—I don't know what it is to be unhappy because I have had to adapt myself—you can call it unhappiness, but I have never had happiness, so I don't know what unhappiness is. Any way, if you see how much pain is in life—ultimate analysis is probably pain any way, isn't it (laughing)? I want to hear you talk now a little, if you talk I will respond (smiling) to your talk (pause). Now, the usual terms, I don't grasp the meaning of terms, there's where feeling comes in—you feel the meaning of terms—you feel the richness and color (pause) in a sunset (pause). Dr. And you have lost that feeling of appreciation? Pt. There is no appreciation—there is no appreciation—no. Dr. What do you think the outcome of your state is? Pt. What the result of it is (pause)? I have forgotten what result means now—what the result of my state is (smiling) (pause). Dr. I mean, do you think you are going to get better or worse? Pt. I think I am gradually shriveling up—going back into childhood (pause); I am quite sure I cannot get better—I am (pause) (yawning drowsily)—no, I don't think it's possible.

For some time the mental condition of the patient varied; he was often dull and sluggish, his motor discharges correspondingly slow and deliberate. He was often silly and childish in bearing. For awhile, in his speech and gait, he simulated an intoxicated person, and always explained his condition as due to his abnormal metabolism which formed alcohol within his system.

The physical examination disclosed the following points of interest. The skin presents a condition of hyperæmic congestion and cyanosis, especially noticeable on the upper back and neck regions. Pressure of hands elicits the so-called X-ray appearance. Dermatographic reflex consists of an intense white, immediate pressure line, with no subsequent red line. Pupils examined in the dark show a somewhat diminished dilatation. With the torch the left pupil reacts about half a millimeter, the right about one and a half, and both very sluggishly. The right is irregular in outline. The accommodative reflex is also diminished in

excursion and time reaction. The tongue is markedly coated and the breath odor is heavy and fœtid. All the deep tendon reflexes in the arms and legs are much increased. The hands and feet are cold and somewhat cyanotic.

During the eight months in which patient has been under observation, there has been considerable physical improvement, with a gain of about 15 pounds in weight. The childish reaction, the intoxication simulation, and the alcohol-formation ideas have largely disappeared. While there is less sluggishness in intrapsychic and psychomotor spheres there is a greater *abaissement du niveau mental* and narrowing of mental horizon and interest.

While patient's insight is still good he is not able to so clearly describe his subjective consciousness as before. In his daily routine he is careless and slovenly in his personal appearance and in his room. His chief interest is his piano playing, and in this he exhibits considerable skill. He keeps much of the time to himself; is sometimes observed to sit and stare in one direction for a half hour, or even more; he has also shown recently unemotional or so-called irrelevant laughter, chiefly at night when alone in his room.

The condition of the skin, pupils and reflexes remain the same as on admission.

Fifty words, chosen with respect to patient's individual characteristics, were used to test the association. The average reaction time of two seconds was prompt, and did not disclose any complex of pathogenic import.

Case II.—Male, single, 27; a Russian-Jew, born of ignorant and illiterate parents. Nothing was discovered of importance in the family history. Patient was the oldest of seven children. He obtained a meager schooling and at an early age was set to work at a sewing machine. He was ambitious to become a lawyer and entered a night school. His lack of preliminary education proved a great obstacle and he failed to pass his final examinations. Three months before admission to the hospital he developed, without sufficient reason, an irritability and impulsivity toward his father, and on several occasions threatened to kill him. He is very reticent when questioned about his father, but patient evidently in some way blames him and his home surroundings for his present situation; in other directions as well, patient has also shown some delusions of paranoid and persecutory character. He was tested with 50 association words—the average time for the whole was 3.9 seconds. The reproduction to the word "father" clearly indicated that a complex had been struck. No reaction was obtained for 23 seconds, then the patient replied "home." He has shown some irritability and impulsiveness toward certain nurses, which, in connection with a frequently occurring refusal to eat, may indicate a hidden paranoid constellation. He walks slowly and holds his body in a strained and awkward position. In conversing he often breaks into a

broad smile without demonstrable reason. The disturbance in the somatopsychic sphere is most prominent and interesting. He spends much of the time on the bed, because when he stands the left side of his chest "jumps so." He repeatedly complains of his heart, fears it is going to stop, says it has no more ambition than he has. Complains of a great variety of paresthesias in surface areas, such as burning, warmth, electric touches, pains and numbness. These often are fleeting in duration, and very localized in extent. Cephalic pain and girdle sensation, he often mentions. He complains of a mental dullness, and that things feel strange to him, or that he cannot think at all and doesn't want to.

In the physical examination the right pupil was more active than the left, and irregular in outline. The excursion of the left under torch-light was diminished. Accommodation reaction is also sluggish and restricted. All the deep tendon reflexes are increased. The extremities are usually cold and moist. There is a very slight grade of cyanosis. In the seven months the patient has been under observation there has been very slow, yet obvious, deterioration, consisting chiefly of affect dementia, an intellectual narrowing and a fixation of the somatopsychic complexes.

Case III.—Male, 27, single. The only son of cultured and intelligent parents. After puberty he evidently showed marked psychopathic predisposition. He was admitted to the Sheppard and Enoch Pratt Hospital in May, 1908. His psychosis was said to have commenced several months previously. The principal feature was the occurrence of mental states characterized by marked disorientation and dissociation in the autopsychic sphere. There were active hallucinations, both visual and auditory. All varieties of delusions of reference paranoid in character were expressed. There were also prominent somatopsychic disturbances, which were, however, at all times present to a lesser extent. Such a phase as the above would last on the average several days to a week, when the auto- and allopsychic disturbance would clear entirely with the return of a good insight and appreciation of having been through some kind of a mental attack. As previously mentioned there would persist, when in his best condition, some somatopsychic disturbance which resembled a neurasthenic reaction. The patient often complained of paresthesias similar in quality to those shown by Case II.

Physical examination was negative except for pupillary anomalies, which were an irregular left pupil and a sluggish light reaction and diminished excursion. Similar attacks to the one described occurred at regular intervals during his stay at the hospital. Between attacks patient's general reaction to the casual observer was practically normal. He was discharged improved, and when heard from recently was able to do some work in the jewelry business, and was considered in good condition by his family. Evidently so far there has been but little deterioration, and if we are right in assuming that the process is a dementing one, the progress is evidently very slow.

CASE IV.—Male, single, 23. Admitted in July, 1905. The psychosis of this patient is strikingly like that of Case III. The attacks, however, have occurred at more regular intervals, and last a week or 10 days, to be followed by a fairly normal interim of about the same duration. The transition into the attack usually takes a few hours. This patient left the hospital in 1907, but has been seen at intervals since then. He shows a well-marked physical and mental deterioration. Formerly an expert stenographer, during his well periods he is still able to do a little work along this line, but the quality of his work has obviously degenerated.

The cyclic-nature of the disturbance in Cases III and IV seem to support the theory of the primary importance of the "X" or toxin of Jung, especially as no complex of pathogenic importance could be discovered in either case.

Cases II and III exhibit peculiar interest from the standpoint of differential diagnosis, inasmuch as a single examination during several phases might easily lead to a diagnosis of neurasthenia. The importance of a correct estimate of such cases is obvious.

THE APPLICATION OF IMMUNITY REACTION TO THE CEREBRO-SPINAL FLUID.*

By J. W. MOORE, M. D.,
Assistant Physician, State Hospital, Central Islip, N. Y.

Time does not allow of a preliminary explanation and discussion of the various theories of immunity by which modern investigators have sought to explain the changes which occur in the serum of an animal which has been inoculated with some foreign cell or toxin.

Suffice it to say that the hypotheses of Ehrlich have so well withstood the assaults of critical experimentation that they have come to be tacitly accepted as, at least, a convenient working basis until chemistry in its rapid strides shall have come to our aid and simplified what are now merely to be expressed as properties possessed by a serum into definite colloid or proteid formulas. In the following paper the use of technical terms has been avoided so far as possible, but such as do occur are among those which were introduced by Ehrlich and are still in constant use.

One of the ingenious applications of an immunity reaction to diagnosis has been the so-called "complement-fixation test" introduced by Bordet and Gengou in 1901.[1] The immune property of a serum, e. g., the immunity which a typhoid patient's serum acquires against the typhoid bacillus, by reason of which it agglutinates and destroys the bacilli in the Widal test, is known to consist of two parts. One part, called antibody, represents that peculiar property of the serum which makes it immune to that organism and to no other. This antibody is thermostabile, that is, it is not affected by heating the serum to 56 C. The other part, complement, is a constituent of all sera whether normal or immune. It is thermolabile and is driven off by heating to 56 C., but can be restored by simply adding a small quantity of fresh, unheated, normal serum from the same or from another suitable species. So, if we first heat the typhoid patient's serum and then add it to an extract made from a broth culture of typhoid bacilli, the organisms will not be destroyed, for the serum has been inacti-

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

vated by the loss of its native complement. But by adding a few drops of serum, from, let us say, a guinea-pig, the patient's serum is at once re-activated and the lysis of the bacilli takes place.

If we have a patient on whom we wish to make a specific diagnosis of typhoid, we have the direct method of demonstrating the bacilli in the patient's blood or excreta, usually a difficult task, or the indirect method of determining the existence of antibody to typhoid in the patient's serum. For the latter procedure we may do the well-known Widal test, or resort to the complement-fixation test, which is somewhat as follows: In a test-tube which we will call tube No. 1, we place a suitable amount of the serum to be tested and of an extract of typhoid bacilli. Next we add a small quantity of fresh guinea-pig serum as complement. If the complement in this guinea-pig serum is absorbed, we can tell that the patient was suffering from typhoid and that an antibody to the typhoid bacillus had formed in his serum, otherwise the complement would remain free, for it is bound only where the serum is specifically immune to the organism present. But there is nothing in the appearance of the fluids in the test-tube to indicate what has taken place; how are we to prove that complement has been absorbed? For this we use, as an indicator, an immune reaction which also requires complement for its fulfillment, but which is easily visible to the naked eye—namely, hemolysis. A rabbit is injected repeatedly with increasing quantities of sheep blood from which the serum has been washed and the corpuscles suspended in physiological salt solution. As a result of this there is generated in the serum of the rabbit an antibody for sheep red-blood cells—a fact which can be demonstrated by bringing the two together, when the suspension of sheep erythrocytes will change in appearance from a bright, opaque red to a clear, transparent cherry color, due to the destruction of the corpuscles and the liberation of their hemoglobin. A small quantity of this rabbit's serum is heated to drive off its complement and placed in a test-tube, which we will call tube No. 2, together with a suspension of sheep red-blood-cells. This gives the potential for hemolysis, complement alone being needed. If the contents of tube No. 1 be added to that of tube No. 2 and hemolysis occurs in the latter, it indicates that free complement existed in tube No. 1 and means that the patient's serum did not contain typhoid antibody. On the other hand, if

hemolysis does not occur, we can say that typhoid antibody did exist in the patient's serum and, acting specifically on the extract of typhoid bacilli, caused the absorption of complement. This is known as the complement-fixation or complement-absorption test.

In syphilis, however, we deal with an organism which has not been cultivated and concerning whose very morphology we are not yet clear. In 1906, Wassermann, Neisser and Bruck[1] published their method of using the complement-fixation test in the diagnosis of syphilis. For an analogue of the extract of typhoid bacilli they used a watery extract of the liver and spleen of a syphilitic fœtus, after first ascertaining by smears that the organs contained large numbers of spirochætes. They added to this the heated serum of a syphilitic and found that complement was absorbed. Conversely, when they used serum from a person who did not have syphilis, complement was not absorbed. Later in the same year Wassermann and Plaut[2] performed the test in general paralysis and tabes, using, instead of the blood-serum, the cerebro-spinal fluid. They obtained the same results as with syphilitic serum, with the same negative controls. The blood serum in these diseases also sometimes gave positive reaction but in a very much smaller per cent than the spinal fluid. Thus it appeared that we were about to be given a new diagnostic criterion for general paralysis and a proof of its specific relationship to syphilis.

It was soon found, however, by other investigators, that an extract from normal livers could be used just as satisfactorily as that from syphilitic organs and that an alcoholic extract did quite as well as a watery one. So it is now known that the presence of syphilitic organisms in the Wassermann extract had nothing to do with the reaction, but that the latter depended upon certain lipoid bodies which are abundant in the liver.

Although the specificity of one element in this so-called Wassermann reaction has been thus exploded, the test remains a valuable one, for a very high percentage of blood-sera from syphilis and spinal fluids from metasyphilitic diseases evince this peculiar property of absorbing complement in the presence of lipoids, while sera and spinal fluids from other disorders are practically always negative in this regard. The sharing of this characteristic by that group of disorders is significant of their relationship.

The technique of the Wassermann reaction as applied to the cerebro-spinal fluid consists in placing a certain quantity (.1 — .2 cc.) of the specimen to be tested in a tube and adding to it the proper amount (1 cc.) of extract of syphilitic or normal organs. 1 cc. of a 10 per cent solution of complement in the shape of guinea-pig serum is then added and the whole incubated at 37 C. for 30 minutes to give time for the complement to be bound. Then the hemolytic system, consisting of 1 cc. of a 5 per cent suspension of red-blood-cells of a sheep and two units, or twice the quantity necessary for complete hemolysis, of the heated serum of a rabbit immunized to sheep erythrocytes, is added. This rabbit serum will cause hemolysis of the sheep blood-cells if there is free complement in the tube, so the tube is again placed in the oven and observed at frequent intervals for several hours. If hemolysis occurs it shows that complement was not bound by the first ingredients and constitutes a negative reaction. The non-occurrence, or blocking, of hemolysis similarly would constitute a positive reaction, since it indicates that complement was absorbed in the first instance. Numerous controls are of course set out for each test.

It will be seen that the process is a laborious one, necessitating as it does the immunizing and bleeding of animals, the making of extracts of organs, the washing of blood-cells, etc. One of the chief objections is that the ingredients are used in a fluid form. In this state they deteriorate steadily and have to be standardized, itself a time-consuming procedure, at nearly every series of experiments. In short, the test was possible only in a well-equipped laboratory and by trained workers.

After a series of experiments, Noguchi[4] of Rockefeller Institute, has succeeded in so modifying and simplifying the technique that it bids fair to become almost a bed-side test. In the first place, he substituted for sheep blood in the hemolytic system, the blood from a human being. Thus, instead of requiring fresh sheep blood for each repetition, it is only necessary to prick the ear or finger of some individual who is known to be free from syphilis, let a few drops fall into a small tube of physiological salt solution, and we have our blood suspension. For the other components in the reaction he has succeeded in impregnating absorbent paper with the liver extract, with the serum of the rabbit which was immunized to human blood and with fresh guinea-pig serum or complement.

Complement, especially, is very unstable and is active only for about three days in the fluid state, while when dried on paper it will retain its strength for months. These various papers are standardized in the laboratory so that small squares of certain dimensions represent, each, the required dose of that particular solution in the test. In paper form the substances will keep for long periods and, of course, are infinitely easier of manipulation. The substitution of the anti-human for the anti-sheep hemolytic system is a great advantage in that it does away with the difficulty which often arises from the presence in human blood-serum of a natural hemolysin for sheep blood.

The possibility of preparing this paper form of the various components in the Wassermann reaction in large laboratories by pharmaceutical houses and its distribution by them, is apparent, and an extremely valuable diagnostic test for syphilis and general paralysis may thus be brought within the reach of any practising physician who possesses a little technical skill.

With regard to the reliability of this reaction in the diagnosis of general paralysis, there have been some widely varying results by different workers. The majority, however, attest to its occurrence in from 60 to 75 per cent of all cases of that disease. In a communication about to be published, Noguchi and the writer have compared the Wassermann reaction with the butyric and other tests as applied to the spinal fluid. The Wassermann reaction, while not giving as high a percentage of positive results as the butyric acid reaction and the cell-count, is of undoubted value, and by eliminating some of the opportunities for error which now exist its efficiency will be much increased. In the series just mentioned, which consisted of about 200 cases, the Wassermann reaction gave 73 per cent of positive findings in general paralysis against 90 per cent which showed the butyric acid reaction and increased cell-content. A negative Wassermann reaction, then, is relatively of much less significance than the other tests, and doubtful reactions are very common. But a definitely positive result can be accepted without much question as meaning a syphilitic or metasyphilitic disorder of the central nervous system. Even in cases of acute inflammatory disease of the meninges in which the proteid and cell-content are very high, the Wassermann reaction is always negative. In two cases of psychoses other than general

paralysis a positive reaction was obtained, but syphilis could not be excluded in their histories. Only four cases of spinal fluid from active lues without nervous involvement were examined, but all were negative. A certain number of such cases, however, have been found in the experience of others to give positive results, and this opens the question of whether these are the persons who, in later life, are prone to develop general paralysis. If such cases could be followed it would help much to clear up the subject of whether the nervous system is invaded at the time of the luetic attack or is subsequently involved.

We may conclude, then, that in general paralysis and tabes, and in lues of the central nervous system, there exists in the cerebro-spinal fluid, besides an increase in cells and proteids, a characteristic which enables it to bind complement in the presence of certain lipoids. That this property is possessed also by syphilitic serum but appears to be absent in other conditions. That the detection of this substance not only aids in the diagnosis of general paralysis, but also is strong presumptive evidence of the relationship of this disease to syphilis.

BIBLIOGRAPHY.

1. Bordet et Gengou: Annales de l'Inst. Pasteur, 1901.
2. A. Wassermann, A. Neisser und C. Bruck: Deutsche med. Wochenschrift, 1906, No. 12.
3. A. Wassermann und F. Plaut: Deutsche med. Wochenschrift, 1906, No. 44.
4. H. Noguchi: Jour. of Experimental Medicine, 1909, March 1.

A REVIEW OF THE RECENT STUDIES OF GENERAL PARESIS.*

By JAMES V. MAY, A. B., M. D.,
Assistant Physician, Binghamton State Hospital, Binghamton, N. Y.

Perhaps nowhere within the wide domain of psychiatry have greater advances been made within the last few years than in our knowledge of general paresis. It constitutes at the present time practically the only psychosis which is accompanied by well-defined characteristic pathological changes which render post-mortem demonstration and diagnosis possible. The gross changes found at autopsy, the thickening and adhesions of the dura, the occasional hemorrhagic membranes and exudates, the excess of cerebro-spinal fluid, the lepto-meningitis with adhesions, the regional atrophies and softenings, the diminished brain weight and the granulations in the ventricles are of themselves usually sufficient for diagnosis. The histological changes which take place have until recently been very vaguely and inadequately described.

For our present comprehensive knowledge of the pathology of paresis we are largely indebted to the exhaustive researches of Nissl and Alzheimer. The latter has laid particular emphasis on the importance of plasma cells in the pia and vessel walls. The pia-arachnoid is always extensively infiltrated by plasma cells and lymphocytes, and shows occasional mast cells. The plasma cells are small and rounded or oval, with usually one and sometimes numerous nuclei. They are specially susceptible to degenerative changes, characterized by vacuolization, with the absorption at times of foreign substances, changes which cause them to resemble granule or waste product cells. Plasma cells are also found in brain syphilis, in tubercular meningitis, in carcinomatous and sarcomatous processes and in encephalitis,—due to lead and other poisons, as well as in idiocy and epilepsy. They occur so much more extensively, however, in paresis as to render a diagnosis comparatively easy, the other conditions in which they occur being

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

excluded by pathological as well as clinical considerations. The presence of plasma cells is readily confirmed by selective stains which have been devised by Unna and Pappenheim. The characteristic meningeal infiltration, although present in practically all areas of the brain, is more marked in the frontal lobes.

In the cortex the vessels seem to be greatly increased in number and show well-marked changes. Aside from an intimal proliferation and a budding of capillaries, there is nearly always an extensive infiltration of the adventitia by plasma cells, lymphocytes, some polynuclear and occasional mast cells. The plasma cells are usually very numerous. Their origin is questionable at the present time. In the superficial layer of the cortex there is almost always an increase of the neuroglia fibrils with numerous spider cells showing eccentric nuclei and large cell bodies. Anglade and Letreille [1] by means of a special stain have shown a conspicuous increase in neuroglia in the molecular layer of the cerebellar cortex. Rod-shaped cells, or " Stabchenzellen " as described by Nissl, with a long nucleus and only slight evidences of a cell body, are very common and seem to have some relation to the severity of the process. Satellite cells and " free nuclei " are often seen in the vicinity of the neurones. The nerve cells are frequently diminished in number in occasional areas with a proliferation of the glia. The definite changes in the neurones which have been described as acute alteration, grave alteration, rarefaction, shrinkage, sclerosis and pigment degeneration are all found in general paresis, but are not pathognomonic of the disease. Many cells are normal and some present the change known as axonal alteration. The cells appear to point in all directions, and there is often an obvious disturbance of the normal layering of the cortex. Areas of softening are often found, but Nissl denies the presence of gummata in paresis. Degeneration of the nerve fibres is shown early in the pyramidal layers and occurs later in the association and projection fields. The various lesions found in the cortex are shown to varying extents in the cord.

The etiology of general paresis has proven as interesting a field of research for recent investigators as that of pathology. The bacterial origin of the disease as advocated by Ford Robertson [2] had led to considerable discussion at times. In cultures from the mucous membrane of the stomach and intestines, from the respira-

tory tract and brains of paretics, he isolated an organism in 1903 which morphologically resembles the Klebs-Löffler bacillus. It was found oftener and more extensively than in genuine cases of diphtheria and was not affected by antitoxin treatment. This has been referred to since as the "bacillus paralyticans." On feeding rats for several weeks with broth cultures of this organism he claims to have found an infiltration of the pia-arachnoid, a proliferation of the neuroglia cells, periarteritis and a degeneration of the nerve cells. In 1907, Robertson stated that the bacillus described "plays the chief part in the product of the toxæmia of general paresis," and reported several cases in which he claimed to have obtained excellent results from the use of anti-bacterial serum prepared in sheep by immunization with the special diphtheroid bacilli. His conclusions have not been generally accepted and the bacillus paralyticans has not been recognized by other pathologists as a factor in the etiology of paresis.

Fairbanks of Boston in 1908[8] reviewed 29 published cases of paresis occurring in children between the ages of 10 and 14 years, the diagnosis having been confirmed in each case by autopsy. Twelve of these cases showed specific stigmata. In 19 cases one or both of the parents presented syphilitic stigmata, or had a definite history of general paresis or syphilis. Of the other 10 cases one had an insane father with syphilitic children, one had several insane relatives, one had normal parents with four other syphilitic children, one had normal parents with one child hydrocephalic and the second and third children still born, one had a brother with evidences of syphilis, two had negative and three unknown histories. He concludes that the infantile cases, which usually have a history of congenital syphilis, point to a specific origin of paresis, and discredit alcohol and other ascribed causes as etiological factors.

The study of the cerebro-spinal fluid by Widal, Sicard and Ravaut, following the introduction of lumbar puncture by Quincke in 1890, has led to interesting results, which have had an important bearing on psychiatry. It was soon found that there was a great increase in the number of lymphocytes in tubercular meningitis, tabes, cerebral syphilis and general paresis. The value of this method of differentiating paresis from other psychoses is readily apparent, and it is at present the most reliable diagnostic pro-

cedure available in doubtful cases. The technique is simple and has recently been improved by Alzheimer so that the sediment, after centrifuging the cerebro-spinal fluid, is imbedded in paraffine, thus permitting much more accurate staining methods. A lymphocytosis is almost universally demonstrable in cases of general paresis. The increased amount of albumen is constant but not of equal importance in diagnosis.

The relation of syphilis to paresis has always been a debated point. According to Paton, Gudden found a definite history of syphilis in 35.7 per cent of his cases, Hirsch in 56 per cent, Jolly in 69 per cent, Mendel in 75 per cent and Alzheimer in 90 per cent. A history of syphilis or evidence of that disease is so often found that I think it safe to say that the majority of observers consider it to be a necessary etiological factor. When the spirochæta pallida was discovered it was hoped that some definite connection between syphilis and paresis could be conclusively demonstrated. Unfortunately this has not been possible and all efforts to isolate the organism in the cerebro-spinal fluid of paretics have failed. A more thorough knowledge of the spirochæta may result in more light on the subject.

A decided step towards the solution of the problem was rendered possible by the application of the principles of the Bordet-Gengou phenomenon,[4] complement fixation in serum diagnosis, to the study of syphilis by Wassermann, Neisser and Bruck in 1906.[5] Serum diagnosis is now thoroughly established and it has been of great value in the study of parasyphilitic diseases in general and paresis in particular. Lesser[6] states that positive reactions are obtained in all cases of paresis. More recent examinations show positive results in over 90 per cent of cases. Marie and Levaditi report similar results. Raviart,[7] after 400 tests in the insane, found positive reactions in paretics with practically no exceptions. The Wassermann reaction would appear to have removed all doubt as to the definite relation between paresis and syphilis.

Efforts to arrive at a simpler method of diagnosis in syphilis and general paresis have resulted in the so-called precipitate reactions. Porges and Meier discovered that a precipitate was formed by the mixture of a .2 per cent lecithin suspension and serum from a syphilitic patient. It was soon found that a similar reaction took place on the addition of solutions of glycocholate or taurocholate

of sodium to luetic sera. The lecithin suspension is made by heating .1 gramme of lecithin in a sufficient amount of alcohol to dissolve it, after which enough distilled water is added to make 50 cc. Glycocholate and taurocholate of sodium are each made up in a 1 per cent solution in distilled water. Equal parts of the serum to be examined and one of the solutions described are allowed to stand for 24 hours in a test tube at the ordinary room temperature. At the end of 15 or 20 hours a precipitate will form in positive cases, varying from a slight flocculence in the upper part of the mixture to a heavy deposit at the bottom of the tube. It becomes quite conspicuous on tapping or lightly shaking the test tube. In some cases it forms more quickly, but should be found in 24 hours at most. Opalescence or turbidity is not sufficient for a diagnosis. The solution should be prepared just before use. Blood serum, either active or inactivated, has usually been used. In the study of syphilitic sera, Butler[8] has found these tests inferior to the Wassermann reaction. Noguchi[9] has found a definite increase of the globulin content in syphilic sera. This is demonstrated as follows: To .1 cc. of spinal fluid add .5 cc. of 10 per cent butyric acid and heat. Then add .1 cc. of a normal solution of sodium hydroxide and reheat briefly. In spinal fluid in cases of general paresis a flocculent precipitate appears. An opalescence is not sufficient. The reaction should take place within two or three hours. Noguchi[9] says in regard to the amounts used in the test, "It is entirely to one's individual taste whether one or a multiple of the quantities indicated above will have to be employed." I have found that in using larger quantities (.5 cc. of spinal fluid, 2.5 cc. of 10 per cent butyric acid and .5 cc. of normal sodium hydroxide) a decided flocculent precipitate is formed in many cases when only a slight or no indication of a precipitate is obtained in using the smaller amounts proposed by Noguchi.

In fluids from cases of general paresis 29 out of 30 examined showed a lymphocytosis; 25 out of 28 gave a positive butyric acid reaction; 17 out of 20 were positive to the glycocholate of sodium; 13 out of 16 to the taurocholate of sodium and 9 which were tested with lecithin all reacted. Six doubtful cases were examined. Of these none showed an increase of lymphocytes, four were positive to the butyric acid test, five to the glycocholate of sodium and five to the taurocholate of sodium. Three of these were tried with

lecithin and all reacted. In the non-paretic cases the butyric acid test gave 14 positive and 11 negative reactions. None of the positive cases had a definite history of syphilis: two of the negative cases are known to have had syphilis. Of the 11 negative cases two cases of dementia præcox, one of alcoholism, one of locomotor ataxia, one of manic-depressive insanity, one of brain tumor and one of epileptic insanity gave a heavy flocculent precipitate after standing from 14 to 18 hours. Two cases of undoubted general paresis reacted only after standing 24 hours. In one case of senility, one of dementia præcox, one of alcoholism, one of epileptic insanity and one of an undistinguished depression, the cerebro-spinal fluid gave a positive butyric acid reaction and a marked lymphocytosis on examination 12 hours after death. In none of these cases was there a history of syphilis or a suspicion of paresis. The significance of a lymphocytosis in post-mortem fluid is not clear. In most of these fluids the sugar had entirely disappeared, although present during lifetime in practically all cases. Glycocholate of sodium was tried in 19 non-paretic cases with 18 positive results. Taurocholate of sodium was positive in 14 out of 15 and lecithin in 13 out of 14 non-paretic cases. The number of positive reactions obtained in non-syphilitic cases would indicate that the precipitate tests cannot properly be considered as specific for either general paresis or syphilis.

To recapitulate briefly, the points I have endeavored to bring out in this paper are the following:

1. The recent researches of Nissl and Alzheimer have resulted in a comprehensive and definite knowledge of the pathological changes which characterize general paresis.

2. Careful investigation has failed to show any definite relation between paresis and the diphtheroid bacillus of Ford Robertson.

3. A lymphocytosis has been definitely established in the cerebro-spinal fluid of paretics and is probably the most reliable diagnostic indication in doubtful cases at the present time.

4. The Wassermann reaction has practically removed all doubt as to the definite relation between paresis and syphilis, although the spirochæta pallida has not yet been demonstrated in the presence of general paresis.

5. While the globulin content of the cerebro-spinal fluid is undoubtedly increased in paresis it does not seem to furnish a basis for a positive diagnosis.

BIBLIOGRAPHY.

1. L'Encéphale, October 25, 1907.
2. Lancet, November 14, 1908.
3. Journal of the American Medical Association, December 5, 1908.
4. Annales de l'Institut Pasteur, 1901, p. 289.
5. Deutsche Medizinische Wochenschrift, 1906, p. 745.
6. Berliner klinische Wochenschrift, September 28, 1908.
7. Revue de Médicine, Paris, September, 1908.
8. New York Medical Journal, October 31, 1908.
9. Journal of Experimental Medicine, January 9, 1909.

A STUDY OF BODY TEMPERATURE IN PARALYTIC DEMENTIA.

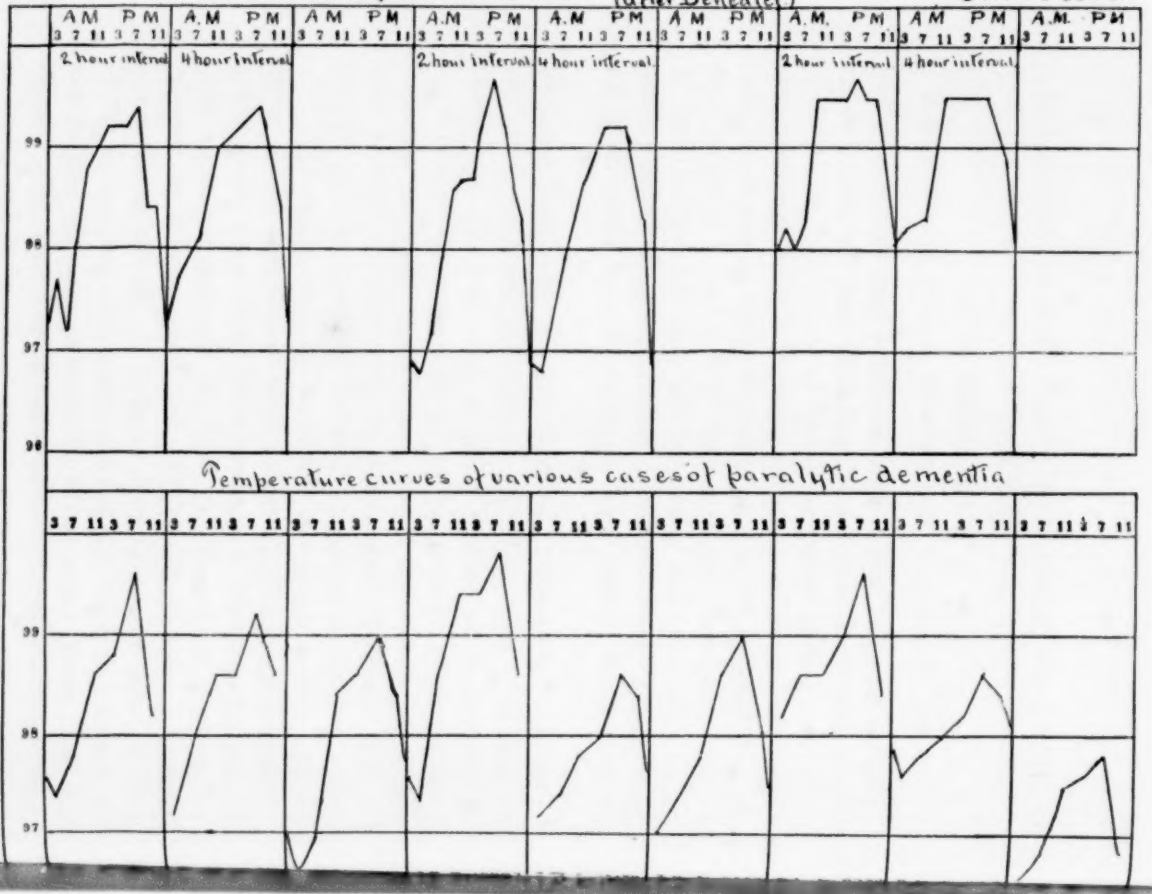
By A. B. COLEBURN, M. D.,

(From the Laboratory of the Connecticut Hospital for the Insane.)

The variations of body temperature have been studied by many writers, and there is universal testimony that the normal individual shows daily variations of .5° to 2° C. (.9° to 3.6° F.). While observers differ as to the point of highest daily temperature and its range, there is an almost complete unanimity of opinion that the lowest temperatures occur between midnight and 6 a. m. No doubt the causes of this daily fluctuation must be apportioned to several different factors—muscular activity, sleep and recumbent position, and ingestion of food being the most active.

The work of Benedict and Snell (1) has determined definitely the amount of heat production resultant on muscular work; the temperature of the subject rising as soon as work was begun, remaining fairly constant during the period of exertion, and dropping during the period of rest; the average rise being about 1° C. The repeated observations of these authors coincide very closely with the experiments recorded by Oberneir (2), Jürgenson (3), Leibermeister and Hoffman (4), and Mosso (5).

Benedict and Snell (1) record three temperature curves which they regard as normal, the records being taken in the rectum every four minutes, night and day, with an electrical resistance thermometer devised by them. The first curve gives a variation of 2.18° F., with a mean average of 98.7° F. In the second curve, the variation is 2.41° F., and the mean daily average 98.34° F. The third curve, taken in the calorimeter while the subject was at rest, is the average of four days and gives a mean daily average of 99° F., and a difference between maximum and minimum of 1.64° F. Chart 1 shows the normal curves given by Benedict, arranged as if temperatures were taken at two- and four-hour


Normal Temperature Curves. (after Benedict)
Chart No. 1.
A.M. P.M.
3 7 11 3 7 11
2 hour interval
4 hour interval
99
98
97
96
Temperature curves of various cases of paralytic dementia
99
98
97

intervals, with specimens of temperature of some of the subjects of this study.

Professor Benedict states that of the large number of temperature curves he has since obtained practically all follow the same variations as those quoted (6).

While mental work has less effect on the body temperature than muscular, Mosso (7) and others have maintained that intense psychical processes may set free so much heat in the brain that its temperature may rise .2° to .4° C. (.36° to .72° F.). Pembrey (8) regards this local rise of temperature as due to vascular changes rather than to mere active combustion in the ganglion cells.

The ingestion of food has a marked effect on body temperature, and in a study of the daily temperature curves given by Pembrey and Benedict it is plainly apparent that the rise occurs when food is being assimilated, and the minimum temperatures occur when the processes of digestion are inactive. It is, of course, to be remembered that the rise is also fairly coincident with the period of the subject's greatest physical activity.

On the other hand, fasting reduces the daily fluctuations of temperature, and so far as experiments show the average temperature is lower.

Sleep in itself has little or no influence on the body temperature. The observations of Leibermeister (4) and others have shown that the temperature of a man asleep does not materially differ from that found at the same time of day when quiet and recumbent. As Pembrey (8) says, "Inactivity causes a fall of temperature, and sleep is a condition in which inactivity is most marked."

Mosso (7), Carter (9), Benedict (1) and Gibson (10) report interesting studies of the results of inversion of the daily routine on the normal temperature curve, their experiments tending to show that while the variation was less in extent, the tendency to persistence of the daily curve was strongly marked.

All observers agree that the temperature in old age is likely to be found equal to or above that of adults. The fœtus before birth has a slightly higher temperature than the mother's womb. At birth the average rectal temperature is 99.5° F., falling shortly to about 98.15° F., then rising within a fortnight to between 99° and 99.7° F., the daily fluctuations being more pronounced than

in adults. The average temperature falls one to two-tenths of a degree from infancy to puberty, and about the same amount from puberty to middle age, thence rising, and about the eightieth year being almost as high as in infancy (8).

No definite agreement between observers has been made as to the influence of the seasons on man, the only constant effect being apparently due to variations in external temperature. Bosanquet (11) states that the highest sustained average rectal temperature occurs during the winter and early spring months.

In 1880 Dr. Mickle, in a work, "General Paralysis of the Insane," as previously reported in various articles on the subject, reiterated the statement that in this disease there is a very definite increase of temperature, stating also that a rise of temperature may accompany a maniacal paroxysm, precede or accompany a convulsive seizure when these are pronounced, may occur without any *apparent* physical or mental cause, may accompany a rapidly progressing case without complications, etc.

Riva (12) confirmed Mickle's observations of rise of temperature during epileptiform attacks in general paresis, and suggested that the (so-called) thermic center is then affected. He (13) later states that in typical cases the temperature is always slightly though irregularly above the normal, and that periods of excitement are always preceded by increase of temperature, though this also may go before stupor or epileptiform attacks. Crömer (14) found that, as a rule, the temperature in paralytic dementia is lower than in health or in other diseases, but that the great differences of temperature occur with the paralytic attacks.

Wirsch (15) found that the temperature was higher during excitement with grandiose ideas, and fell when the mood became calmer, as a rule, but not all maniacal attacks raised the body temperature.

Rottenbiller (16) gave results of 4724 observations of temperature in 33 cases of paralytic dementia, deducing that the prevailing temperature is subnormal, that great daily variations frequently occur without apparent cause, and that these characteristics are present in the early stages of the disease and in remissions, and are consequently of diagnostic value.

Dr. Savage (17) opined that in dementia paralytica there was little increase in the earlier stages, but in the later stages, besides

recognizing the influence of "fits," stated that in uncomplicated cases the morning temperature was about 100° and the evening 101° to 102° F.

Crichton Browne (18), in a series of observations on insane patients, found that cases of paralytic dementia gave the highest mean temperatures.

Peterson and Langdon (19) report observations on 25 cases of paralytic dementia for one week, concluding that unusual variations of temperature in these cases were not due to conditions related to the pathological phenomena of the disease, but depended on thermogenic features not recognized by the observer (not denying the occurrence of rise of temperature with paralytic and convulsive attacks), and that the average bodily temperature and daily oscillations were practically normal. Parsons (20) gives a detailed study of a case where the rise of temperature and convulsions seemed wholly independent of each other.

Sorokovekoff (21) concludes that the elevated temperatures in general paresis occur in the maniacal forms; that the elevated temperatures are not wholly due to physical causes, but are often accompanied by phenomena plainly due to excitation of the central nervous system; that the periods of temperature approaching normal are found in the demented forms, depressed and stuporous states.

The writer for some years past has made temperature observations on cases of paralytic dementia in the Connecticut Hospital for the Insane for periods of from a few weeks to over two years continuously, the temperatures being recorded every four hours of the day and night. The diagnosis in all but a few of the cases observed was confirmed by autopsy findings, and of these few no question of diagnosis was ever raised.

The difficulties and annoyance to the patient, resultant on taking the temperature in the rectum or mouth, led to the adoption of the axilla, and comparison of the axillary temperatures on the two sides showed the variations to be slight and inconstant. In most cases, the normal temperature curve was fairly evident (though the high point was often later than given by Benedict), and in only a few cases was there inversion; this more often occurring during the later stages of the disease.

In the two cases tabulated together for comparison in Table 1, the mental disease was fairly well advanced, but there was no marked evidence of meningeal irritation, nor did there occur either paralytic or epileptiform attacks. The extremes of variation were .6° and 2.8° in the case of W. F. H., the average variation being 1.56°, and .6° and 2.4° in the case of J. R., the average being 1.41°.

TABLE 1.

DEGREES FAHRENHEIT.

	W. F. H.			J. R.		
	Max. Tempt.	Min. Tempt.	Range.	Max. Tempt.	Min. Tempt.	Range.
Feb. 1.....	97.8	95.0	2.8	99.8	97.4	2.4
2.....	97.8	96.8	1.0	99.0	97.7	1.3
3.....	97.6	96.4	1.2	97.8	97.0	.8
4.....	98.0	96.8	1.2	98.0	97.2	.8
5.....	98.2	96.6	1.6	98.2	97.0	1.2
6.....	98.2	97.0	1.2	98.4	97.1	1.3
7.....	98.4	97.8	.6	98.0	96.8	1.2
8.....	97.8	96.6	1.2	98.4	96.6	1.8
9.....	98.6	97.0	1.6	98.4	97.0	1.4
10.....	98.4	96.8	1.6	98.6	96.8	1.8
11.....	98.0	96.8	1.2	98.4	96.8	1.6
12.....	98.0	96.6	1.4	99.0	97.0	2.0
13.....	98.4	96.8	1.6	98.4	97.0	1.4
14.....	98.2	96.9	1.3	98.6	97.2	1.4
15.....	98.4	96.7	1.7	98.0	97.4	.6
16.....	99.0	96.8	2.2	98.4	96.6	1.8
17.....	98.0	96.0	2.0	98.4	96.6	1.8
18.....	98.6	97.0	1.6	98.6	97.4	1.2
19.....	98.8	96.6	2.2	98.7	96.9	1.8
20.....	98.8	97.8	1.0	98.6	96.6	2.0
21.....	98.4	96.7	1.7	98.2	97.6	.6
22.....	97.2	96.0	1.2	98.0	97.0	1.0
23.....	97.5	95.4	2.1	98.6	96.6	2.0
24.....	97.2	95.3	1.9	98.4	97.6	.8
25.....	97.4	96.2	1.2	98.4	97.0	1.4
26.....	97.6	96.0	1.6	98.4	97.2	1.2
27.....	98.0	95.8	2.2	98.4	96.8	1.6
28.....	98.2	96.6	1.6	98.4	97.0	1.4
Ave.....			1.56	Ave.....		1.41

W. F. H. was of the demented form of general paresis, poorly nourished, inco-ordinate and confined to bed.

J. R. was of the expansive type, excellently nourished; incoordinate; kept in bed, but more active and restless than W. F. H.

These two cases were in the same ward and under the same external influences. Both were moderately active within a limited range, but one was silent and the other constantly babbling of his great possessions. The temperature of the ward varied between 62° and 72° F., but most of the time was about 68°. At the hours in the early morning when W. F. H showed temperatures below 96° F. on the 23d, 24th and 25th, the ward temperature was 66° or 67°. The 95° temperature on the first was taken shortly after the patient had been bathed and the ward thermometer stood at 72°.

J. M. (Chart 1).—In the case of another paretic on whom records of temperature were made for over a year, the daily temperature for a week or more at a time would not rise above 98.4° or fall below 97°, the average daily variation being less than 1°, and the daily rhythm was almost typical, though the range was low and limited.

E. L. (Chart 1), for five months prior to his death, in a series of over 900 observations, showed a temperature below 97° only five times, the lowest being 96.2° and going to or above 100° four times, the highest temperature being 100.4°, the record lying between 97.8° and 98.8° in the great majority of instances.

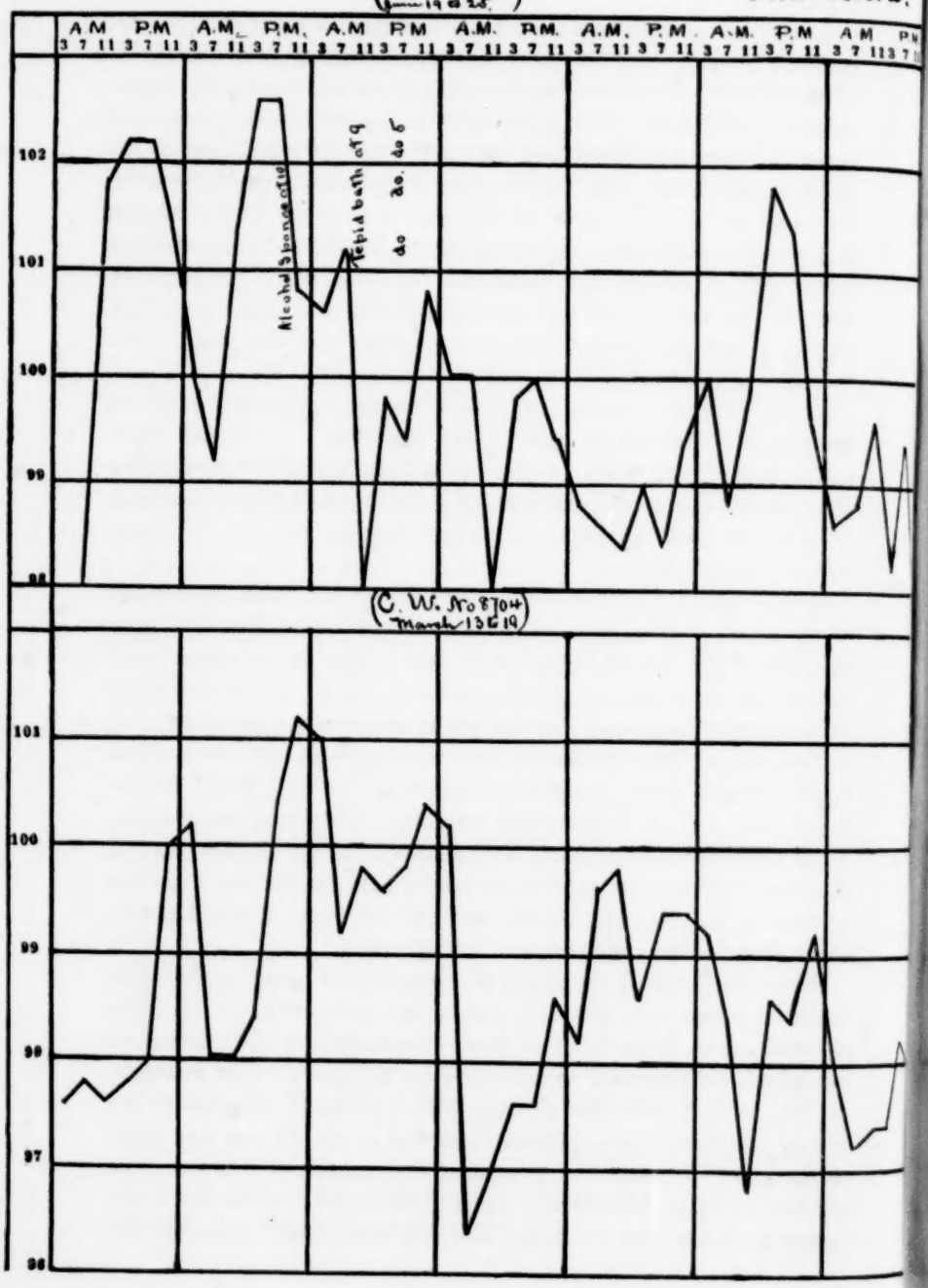
The autopsy showed chronic leptomeningitis in both these cases. J. M. showed high, variable temperature for five weeks before death, but E. L.'s temperature remained subnormal until death.

All these cases were fairly well advanced in the disease, and if, as some writers have asserted, increased heat production is always present in this disease, should have shown more marked differences from normal temperature variations.

While the marked variations of temperature noted in the cases under consideration could in many cases be ascribed to some physical cause, there were at times exacerbations of temperature for which no plausible cause could be assigned. For example, W. W., male, a demented paretic, with a probable duration of the disease of three years, suddenly showed a rise in one day from 98° to 102.2° F., followed by violent fluctuations of this sort lasting for 10 days (Chart 2). The rhythm was of normal type, except as influenced by baths, etc. A week later a similar rise

(W.W. No 10333)
June 19 to 25.

Chart No. 2.



occurred, but the height was less and the fluctuations of more limited extent, though three weeks elapsed before the daily variation became less than 2°. Four months later, when rather brighter mentally, for three weeks continuously, this patient's temperature remained between 97.6° and 98.8°, the daily variation never exceeding .6° F.

Another case, C. W., female, of similar form and duration to the previous case, frequently showed rapid and unaccountable variations, sometimes nearly 4° in a day, at irregular intervals, with intervening periods when the range was normal or less (Chart 2).

Other patients would from time to time, for a few days or a week, show temperature of a higher or lower plane than normal, but of ordinary range and curve.

In all these cases it seemed possible, at the time these particular changes occurred, to exclude all acute inflammatory conditions or other physical causes.

In about one-quarter of the cases under consideration the blood was examined microscopically, but without any positive relation being established between the height of the temperature and the changes in the blood, independent of meningitis or other acute inflammatory conditions.

In most cases the percentage of hæmoglobin was low, but in only two cases fell below 72 per cent, one case on one occasion showing a drop in hæmoglobin from 95 to 65 per cent in six days, with a practically normal temperature, and dropping to the same percentage on two other examinations from 80 and 84 per cent, the examinations being made at intervals of about a week and at the same time of day. This patient was active and well nourished at this time and had no acute ailment.

The other case dropped from 78 to 58 per cent in two weeks with the advent of an acute attack of leptomeningitis, with great restlessness and loss of weight.

The leucocytes were increased in every case examined at some time in the disease, but in these cases it could be ascribed either to meningitis, acute decubitus, pneumonia or other acute inflammation. The highest number to a cubic millimeter was 27,000, reached by two leptomeningitis cases. The proportion of polymorphonuclear neutrophiles was increased regularly, reaching 94.8

per cent in the case of A. B., and 91 per cent in J. M. two days before death from bronchopneumonia.

This observation as to the pathologic increase in "polynuclears" in the terminal stage of paralytic dementia confirms the report of Diefendorf (22) on blood changes in dementia paralytica. An interesting observation was that the percentage of hæmoglobin in the emaciated cases was not appreciably lower than in the well-nourished cases.

Cases A. B. and A. G., both of which gave physical symptoms of meningitis, confirmed by autopsy, are given for comparison (Chart 3). Both men were almost completely paralyzed, could swallow only small quantities of fluid food, showed wide ranges of temperature, and both had been losing in weight for months. The temperature of A. B., the better nourished of the two, during the last week of his life, ranged from 98° (once 97.2°) to 104°, while A. G. (slight tubercular infection) did not during that time show a temperature above 99°, it being most of the time about 95°, and showing an inversion of rhythm.

In three cases the writer has tabulated the blood examinations for the last months of life.

The percentage of hæmoglobin in the case of A. G. (Table 2) had been high prior to an exacerbation of the meningeal symptoms, running sometimes even higher than the scale, coincidently with an increase in red cells and due probably to concentration of the blood, but the temperature had fluctuated widely and risen to 100° and over before the hæmoglobin became markedly decreased. The leucocytosis, which had been marked earlier in the observations, diminished toward the close of life (insufficient nourishment), while the polynuclear percentage remained high throughout. Before the temperature became subnormal, while still reaching 101° at times, the erythrocytes had dropped below 4,000,000, and three days before death rose to 4,670,000, with a temperature ranging between 95.2° and 97.8°.

In the case of J. A. K. (Table 3), who had four convulsive attacks during the observations, it was found that in two attacks the leucocytes were increased and in two they were diminished as compared with the immediately previous observation.

In the case of E. L. (Table 4) the temperature and leucocytes

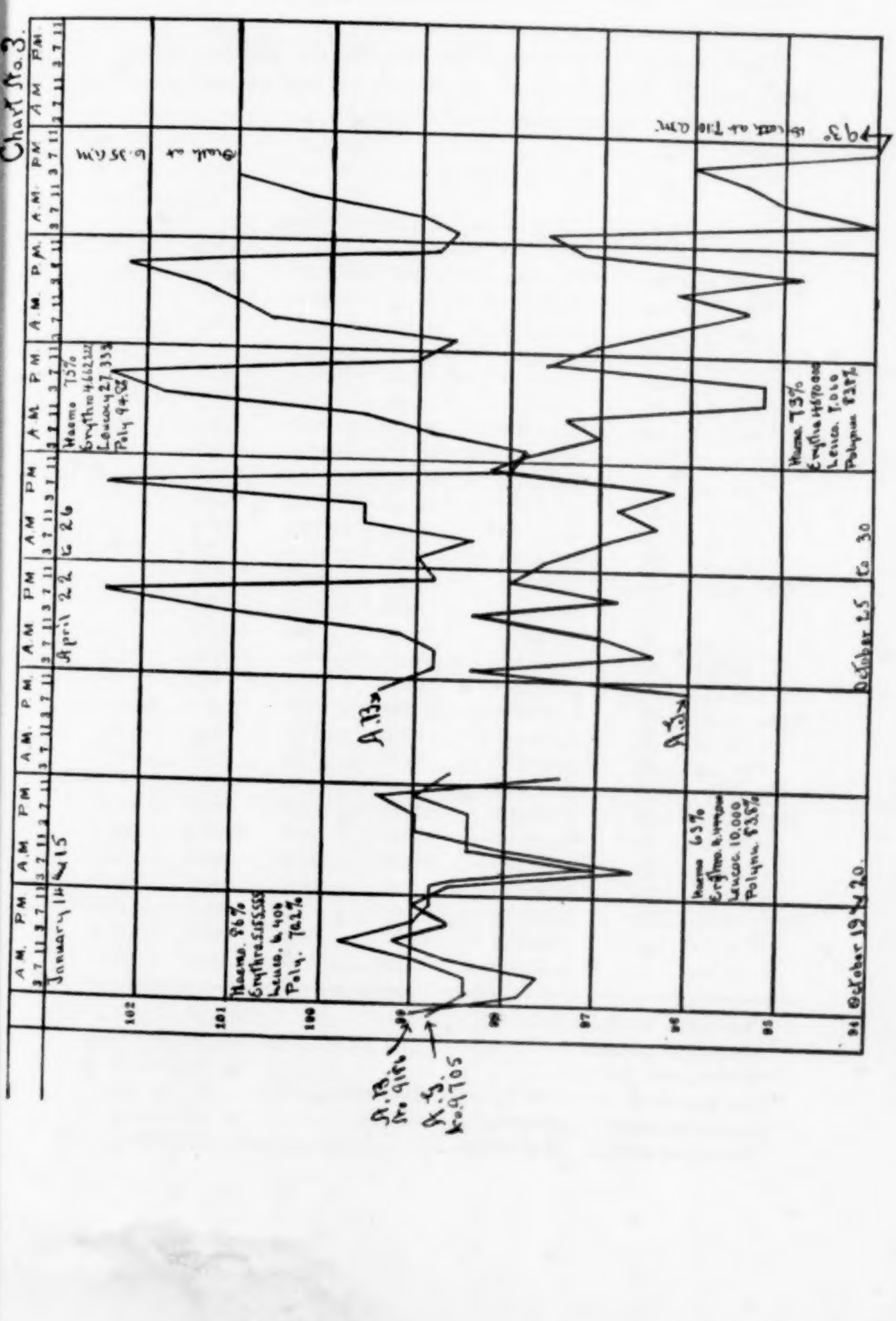


Chart No. 3.

remained practically normal throughout, although the percentage of polynuclears increased above normal.

There seemed little connection between the highest temperatures and the leucocytosis, as in only about half of the instances were they even approximately coincident.

TABLE 2. A. G.

		Hæmoglobin %.	Erythrocytes.	Leucocytes.	Polynuclear %.	Temperature (Fahr.)	
						High.	Low.
Dec.	16[1]	96	6,111,111	14,666	84.6	98.4	96.4
	23	100	6,480,000	15,944	83.2	98.5	96.4
	30	90	6,258,888	10,400	72.6	98.5	96.6
Jan.	6	98	5,960,000	12,066	78.8	98.2	96.6
	13	102	6,177,777	17,500	82.4	98.6	97.6
	20	100	6,355,555	12,466	76.8	98.6	97.2
	27	95	6,058,888	8,000	76.2	99.0	97.2
Feb.	3[2]	105	6,031,111	10,666	80.9	99.0	96.0
	10	96	5,509,000	11,600	75.0	98.0	96.4
	19[3]	97	5,808,888	10,066	81.2	98.8	98.8
	24	95	4,875,555	12,700	83.9	98.4	97.6
	28	85	5,622,222	15,500	82.0	98.6	97.4
Mar.	3	65	5,633,333	11,933	83.2	98.8	97.2
	10	83	5,562,444	9,466	80.8	98.8	97.4
	17	77	5,768,888	13,333	77.8	98.3	97.5
	24[4]	90	6,188,888	18,533	88.2	99.4	97.8
	31[5]	80	5,506,666	10,216	76.6	98.4	97.2
Apr.	7	87	4,818,888	10,600	81.8	98.0	97.2
	14	85	5,844,444	12,110	85.6	99.6	98.0
	21	82	5,888,888	11,066	82.6	99.8	98.8
	28[6]	80	5,426,666	16,066	86.0	100.2	98.0
May	5	98	5,293,333	11,000	85.6	98.8	97.6
	12	77	5,257,777	12,000	76.6	99.0	98.4
	20	80	5,377,777	17,666	85.8	101.6	98.0
	26	74	5,071,111	14,000	80.6	101.0	97.8
June	2	80	5,051,777	15,450	77.6	101.2	98.6
	9	93	5,626,666	20,666	80.4	98.8	98.2
	16	75	5,373,333	14,333	85.8	101.0	97.6

[1] Emaciated, weak, in bed.
[2] Weight, 98 pounds.
[3] Weight, 90¼ pounds.
[4] Day preceding, short rise to 103°; dysenteric attack.
[5] Convalescent from dysentery.
[6] Gaining in weight. Bed-ridden.

TABLE 2. A. G.—CONTINUED.

	Hæmoglobin %.	Erythrocytes.	Leucocytes.	Polynuclear %.	Temperature (Fahr.)	
					High.	Low.
June 23[7]	85	5,937,777	14,666	85.8	99.0	97.6
30[8]	83	5,013,333	13,733	88.2	99.4	98.0
July 7	77	5,388,888	19,867	85.4	99.8	98.4
14	70	4,786,666	20,200	86.8	101.0	97.8
21	78	5,448,888	20,133	83.0	98.8	97.0
28	67	4,791,111	14,933	89.4	100.8	98.6
Aug. 4	58	4,782,222	10,600	84.2	101.0	97.0
11[9]	65	4,813,333	10,733	82.8	99.4	97.2
18	63	4,880,000	21,266	84.4	101.4	98.6
25	60	4,644,444	12,400	84.8	99.6	98.4
Sept. 1[10]	73	4,964,444	12,933	83.6	105.0	98.8
8	65	4,768,888	15,133	85.0	99.0	98.6
15	65	4,351,111	17,733	85.0	99.2	98.6
22	63	3,764,444	15,900	84.2	101.2	98.6
29	60	4,360,000	14,000	85.8	100.8	97.0
Oct. 6	70	3,770,000	13,600	85.2	100.2	97.8
13[11]	69	3,888,800	11,700	85.0	101.0	98.0
20	63	4,444,000	10,000	83.8	99.0	97.0
27[12]	73	4,670,000	7,060	83.8	97.8	95.2

TABLE 3. J. A. K.

	Hæmoglobin %.	Erythrocytes.	Leucocytes.	Polynuclear %.	Temperature (Fahr.)	
					High.	Low.
Dec. 13	95	5,586,666	14,066	88.6	99.0	——
20	85	5,413,333	19,733	80.6	101.0	97.2
28	90	5,413,333	11,333	79.4	99.0	97.6
Jan. 4	95	5,222,222	14,210	79.2	99.0	97.8
10	88	5,328,888	6,533	76.0	99.2	98.2
17	88	6,293,333	7,266	67.8	99.0	97.0
24	95	5,671,111	11,533	64.2	99.2	97.2
31	97	5,964,444	10,133	73.6	99.2	97.4
Feb. 7	106	6,325,555	10,933	80.4	98.8	97.4
14	102	5,874,444	9,466	72.4	98.8	97.8

[7] Alternating stupor and animation.
[8] Losing in weight.
[9] Restless, rotating head, noisy, losing flesh.
[10] Rocking head and moaning.
[11] Small quantities of fluid food only.
[12] Died October 30. Autopsy showed tubercle in lung, spleen and mesenteric glands. Chn. Leptomeningitis.

TABLE 3. J. A. K.—CONTINUED.

		Hæmoglobin %.	Erythrocytes.	Leucocytes.	Polynuclear %.	Temperature (Fahr.)	
						High.	Low.
Feb.	21	80	5,146,666	7,733	70.0	99.0	97.0
	28	102	6,013,333	10,333	60.6	98.8	98.0
Mar.	7	72	5,393,333	5,000	78.6	98.8	98.0
	13	76	4,977,777	7,066	80.0	98.2	98.0
	21	78	5,071,111	9,533	81.0	99.2	97.6
	28[1]	80	5,511,111	10,533	77.6	98.8	98.0
Apr.	4	84	5,364,444	8,933	65.2	99.0	98.0
	11	86	5,217,777	5,333	68.6	98.8	98.0
	18	90	5,477,777	5,933	76.0	102.0	98.2
	25	82	5,551,111	6,400	74.8	98.8	98.0
	30[2]	75	4,777,777	13,950	90.4	100.0	98.2
May	2	85	5,337,777	14,650	86.2	100.2	97.0
	9	80	4,942,222	9,466	74.0	99.2	98.0
	16	78	5,422,222	7,400	75.6	100.4	97.8
	23[3]	77	5,372,222	4,600	77.2	99.6	98.6
	30	80	5,322,222	11,666	74.0	98.8	97.0
June	6	76	4,995,555	15,000	71.2	99.0	98.0
	13	70	4,915,555	5,966	74.8	102.8	98.4
	20	75	4,315,555	6,133	64.0	99.0	98.0
	27	82	5,191,111	7,133	84.0	98.8	97.8
July	5	85	5,017,777	6,000	70.8	98.8	98.0
	11	88	5,493,333	9,867	76.4	98.8	97.0
	18	72	4,810,000	6,400	74.0	98.8	98.0
	25	67	4,551,111	10,200	72.6	99.0	97.8
Aug.	1	65	4,666,666	9,200	74.0	99.0	98.2
	8	80	4,897,777	6,200	77.8	99.0	98.0
	15	80	5,071,111	6,866	67.0	98.6	97.0
	22	73	4,693,333	10,333	72.2	99.0	98.0
	29[4]	82	5,546,667	7,750	76.4	100.0	96.4
Sept.	5	73	4,533,333	8,666	76.6	99.0	97.8
	12[5]	80	4,700,000	10,000	87.4	100.8	99.4
	18[6]	78	4,131,111	13,733	84.8	100.8	99.6
	22[7]	—	3,600,000	10,700	——	105.0	102.8

[1] Paralytic attack. Temperature 97.8°, pulse 136, respiration 20. Blood examined an hour later.

[2] Convulsions day previous; temperature 100.8°, conscious.

[3] Day previous, temperature 104.4°.

[4] Convulsion August 27.

[5] Convulsion.

[6] Pneumonia.

[7] Died September 23. Autopsy showed leptomeningitis and pneumonia.

TABLE 4. E. L.

		Hæmoglobin %.	Erythrocytes.	Leucocytes.	Polynuclear %.	Temperature. (Fahr.)	
						High.	Low.
Feb.	13[1]	95	4,804,444	6,266	74.0	99.0	98.4
	20	68	3,920,000	5,333	83.8	98.6	97.0
	27	75	5,020,000	9,533	81.8	98.8	98.0
Mar.	6	83	5,213,333	6,200	72.0	99.4	97.0
	13	65	4,706,666	7,733	80.6	99.2	97.8
	20	76	5,208,888	6,133	68.2	98.2	97.4
	27[2]	85	5,915,555	6,250	82.8	98.6	98.0
Apr.	3	82	5,546,666	9,433	73.4	98.4	97.4
	10	70	5,824,444	7,733	79.0	98.2	97.2
	17	76	5,264,444	5,000	86.0	98.2	96.6
	24	70	5,271,111	7,777	77.8	98.2	96.6
May	8	80	5,533,333	7,866	74.2	98.6	97.8
	24[3]	68	4,862,222	6,000	76.0	98.6	97.6
June	5	75	4,746,666	8,866	87.7	98.2	97.4
	19	80	5,328,888	7,266	81.4	100.0	97.8
July	2[4]	78	5,333,333	8,800	77.8	98.6	97.8

The body temperature in the later stages of paresis is unquestionably affected by the condition of the digestive system. In fully half of the cases, during this observation at some time, an otherwise unexplained rise of temperature, with a history of constipation for two or more days, was brought back to a normal range by a cathartic or enema.

The influence of fasting on the temperature has been studied, chiefly in the lower animals, with uniform results, the temperature dropping markedly till death ensues. Noyes (23) records the temperature of 94° in a demented man who had taken no food for 45 days. Three of the cases observed by the writer, where paralysis prevented swallowing, ran temperatures below 95° prior to death. Other similar cases, but with a terminal pneumonia, showed abnormally high temperatures.

In six cases, paralytic and epileptiform attacks occurred once or oftener during the period of observation, and in five there was

[1] Weight, 158 pounds.
[2] Weight, 146 pounds.
[3] Weight, 137 pounds.
[4] Died July 8. Convulsion, no rise of temperature.

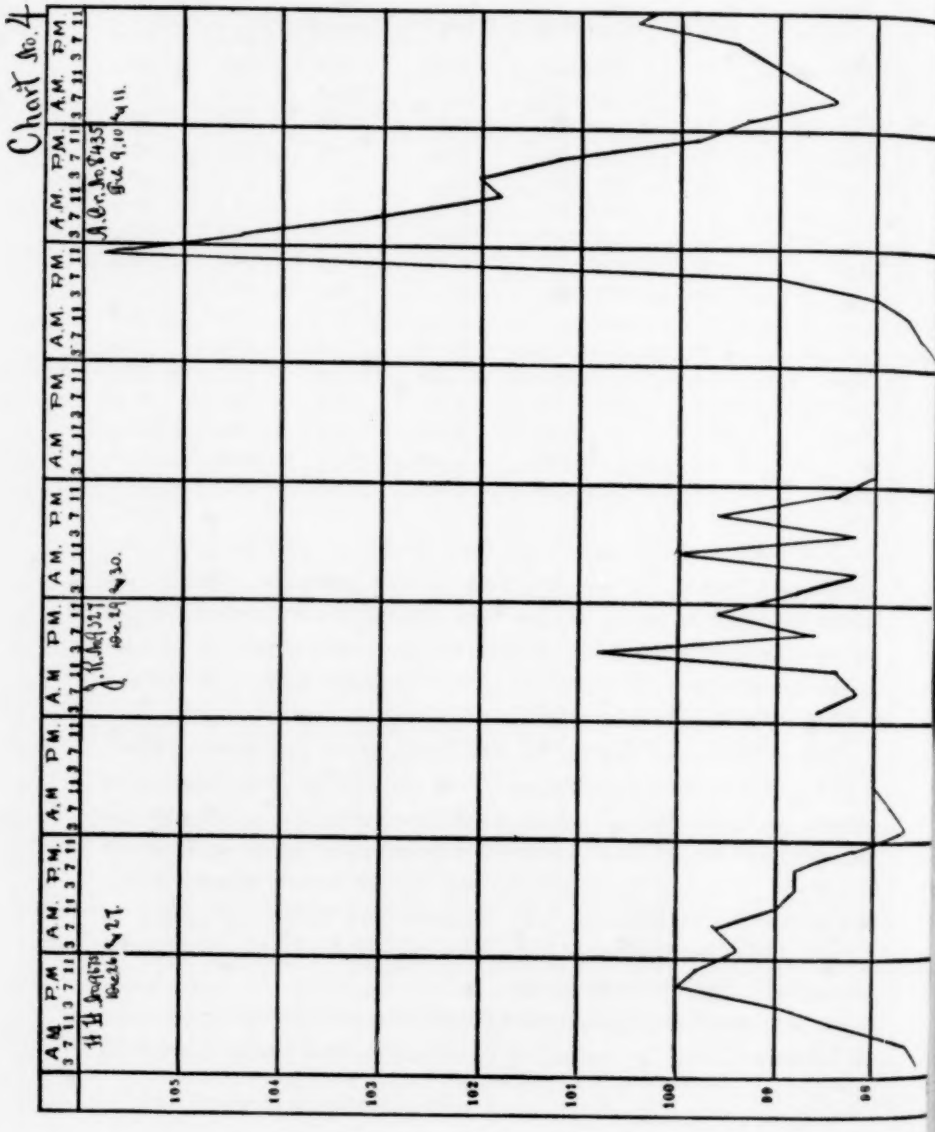


Chart No. 4
A.M. P.M. 3 7 11 3 7 11 3 7 11 3 7 11 3 7 11 3 7 11 3 7 11 3 7 11 3 7 11 3 7 11
105
104
103
102
101
100
99
98

a rise of temperature which could be attributed to the attack (Chart 4).

In Chart 4 are temperature curves of three cases. The first two cases had been previously ranging between 97.6° and 99° for weeks. The third had shown a wider range, but a fairly normal rhythm. In the first case (H. H.) there were two convulsive attacks two hours apart, stupor for four hours and a transient paralysis of the right side. In the second case, J. K., there was no loss of consciousness, but a convulsion of the whole left side, continuing at intervals for several days, and similar attacks occurred during the next six months. In the third case there was a general convulsion with loss of consciousness, stupor for several hours and spasm of the glottis, when swallowing was attempted, for two days. These patients had had no seizures prior to those here charted, and in all the disease was of the expansive form. In the other patient the attacks were milder in character, and while loss of motion and consciousness occurred, there was little or no convulsion.

Two patients who were subject to attacks of excitement and furor regularly showed a sharp rise of temperature when a thermometer could be used (Chart 5, J. M.).

As with animals in which the spinal cord has been cut, the body temperature can be more easily lowered or raised by external agencies, so in many cases of paralytic dementia the effect of a cool bath or of hot, humid atmosphere is distinctly greater in depressing or elevating the temperature than with other patients. It is not an uncommon experience with the writer to find, after a hot afternoon, especially if the air is loaded with moisture, that on a ward with, say 20 cases of paresis, three or four will have developed temperatures ranging above 100.5° F. These are, as a rule, patients in the later stages of the disease, bed-ridden, inco-ordinate in muscular action, obese and often showing trophic changes in the skin. The high temperature is, as a rule, quickly relieved by cool baths or sponging, but not infrequently has a distinct effect in causing collapse in patients nearing their demise (Chart 2, W. W.).

The degeneration of the vasomotor nerves, with their center in the medulla, is probably in a large measure responsible for the loss of power of heat regulation, but that the vasomotor nerves

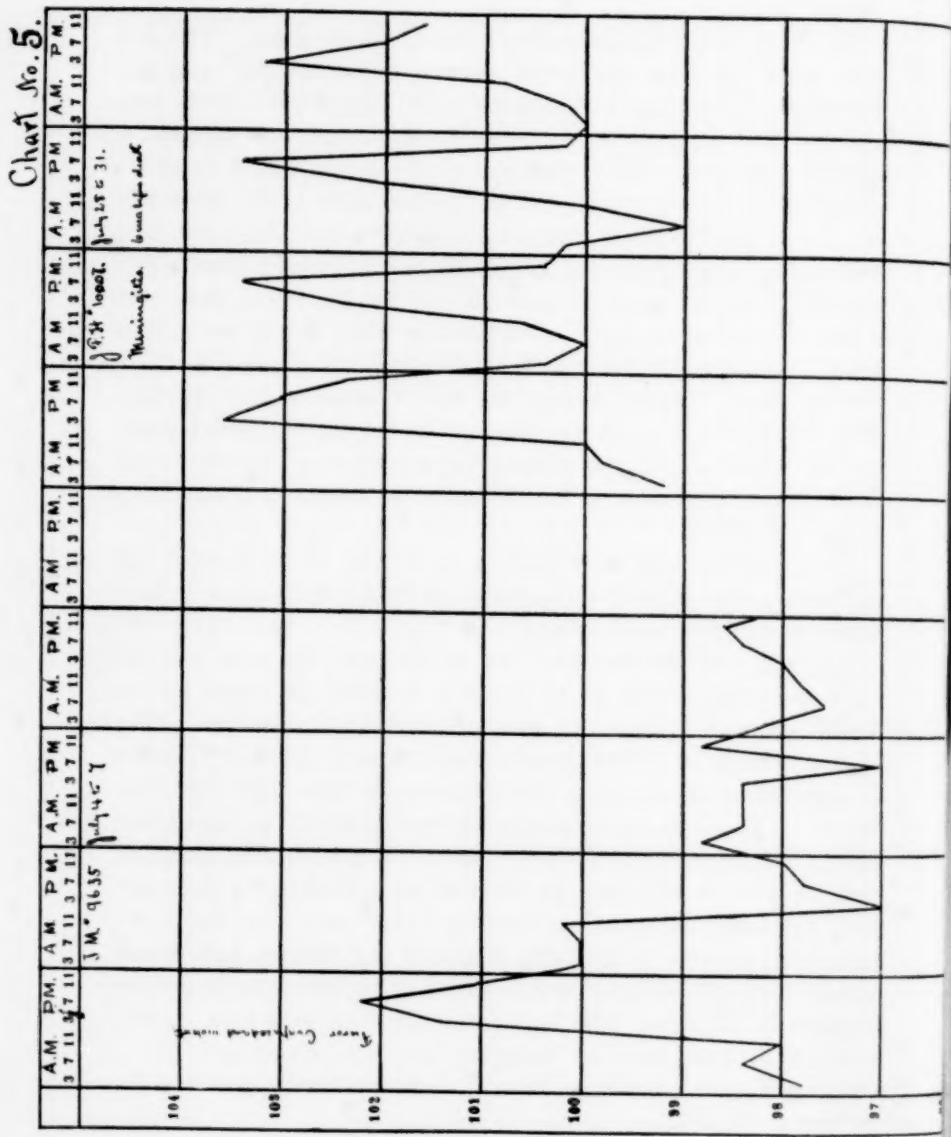


Chart No. 5.
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
A.M. P.M.
3 7 11 3 7 11
104
103
102
101
100
99
98
97

have subordinate centers in the spinal cord is well established, and it seems probable that the degenerative changes in the sensory roots of the spinal nerves and the cord may in some cases involve these centers. The existence of heat centers in the brain is not yet beyond question, though the last observations the writer has found (24) seem to establish their existence in the higher warm-blooded animals.

In the 31 cases of the present study, when high temperatures occurred with evident meningeal irritation, while the range of temperature might be very large, the rhythm was much more regular than in the cases apparently due to furor, minor physical ailments, external causes of discomfort, etc. The temperature in the first set of cases might range between 98° and 104°, as in the other cases, but had not the sudden onset, kept its general character for weeks and usually the temperature curve resembled the normal in spite of the wide range (Chart 5).

Conclusions.

The cases observed early in the disease showed no appreciable departure from normal in temperature. The curve was influenced by the time of day, ingestion of food, sleep and exercise as in healthy individuals.

When the disease had progressed further, anger, excitement, epileptiform and paralytic attacks, minor physical ailments, etc., produced effects on the temperature out of proportion to the causes, while some elevations of temperature unaccompanied by leucocytosis occurred for which no physical basis could be assigned.

The inflammatory changes in the brain and spinal cord apparently are largely causative of the high temperatures occurring with leucocytosis.

On the whole, the general results of these observations confirm the opinions of Rottenbiller (16), except that the writer cannot fully accept his conclusions as to the diagnostic value of the temperature variations.

Sorokovekoff's conclusion that elevated temperatures in paresis are often due to excitation of the central nervous system would seem to be borne out by this study, but the demented state, while lower in average than the maniacal, gave no nearer approach to normal curves.

BIBLIOGRAPHY.

1. Benedict and Snell: Archiv für die gesammte Physiologie, 1901, Vol. LXXXVIII, p. 492; 1902, Vol. XC, pp. 33-72.
   Benedict: Amer. Journ. of Physiol., 1904, Vol. XI, pp. 145-169.
2. Oberneir: "Der Hitzschlag" Bonn, 1867.
3. Jürgenson: Der Körperwarme des gesunden Menschen, Leipzig, 1873.
4. Leibermeister and Hoffman: Handbuch d. Path. u. Therap. d. Feibers, Leipzig, 1875.
5. Mosso: Vergleichende Phys. der haussäugethire, 1892.
6. Myers: (Quoted from Benedict.) Yale Med. Journ.
7. Mosso: Die Temperature des Gehirns, Leipzig, 1894.
8. Pembrey: Schäfer's Physiology, London, 1898.
9. Carter: Journ. Nerv. and Ment. Dis., Vol. XVII, p. 785, 1890.
10. Gibson: Journ. Med. Sci., 1905, Vol. CXXIX, p. 1049.
11. Bosanquet: "Annual Heat," Todd's Cyclopædia, Vol. II, p. 659.
12. Riva: Rivista Sperimentale, 1879.
13. Riva: Arch. Soc. Freniatrica, 1883.
14. Croemer: Allg. Zeitschr. für Psych., Vol. XXXVI, Band 2, u. 3 Heft.
15. Wirsch: Centralblatt für Nervenheilkunde, Mar. 1, 1881.
16. Rottenbiller: Centralblatt für Nervenheilkunde, Jan. 1, 1889.
17. Savage: Quoted by Turner, Journ. Ment. Sci., Vol. XXXV, p. 347.
18. Crichton Browne: Quoted by Turner, loc. cit.
19. Peterson and Langdon: Journ. Nerv. and Ment. Dis., Vol. XVIII, p. 750, 1893.
20. Parsons: Journ. Nerv. and Ment. Dis., 1895, Vol. XX, p. 407.
21. Sorokovekoff: Moniteur neurologique, Rus., 1904.
22. Diefendorf: Trans. Amer. Med. Psy. Assoc., 1903.
23. Noyes: Brit. Med. Journ., Vol. II, p. 551.
24. Ott: Jour. Nerv. and Ment. Dis., 1893, Vol. XVIII, p. 774.
    Ott and Scott: Jour. Exp. Med., 1907, Vol. IX, p. 61; Tangl. Pflüger's Arch., 1907.

THE VALUE OF STAFF CONFERENCES IN STATE HOSPITALS.*

By ELBERT M. SOMERS, M. D.,

First Assistant Physician, St. Lawrence State Hospital, Ogdensburg, N. Y.

Staff conferences, essentially medical in character, have been in operation in the various State hospitals of New York State since the year 1905, as required by law.

The introduction and adoption of newer methods of clinical study, shortly before this period, clearly emphasized the need of more uniform and concerted action on the part of hospital staffs with reference to the study of the mental and physical condition of those admitted for care and treatment.

Previously, some of the hospitals had, for some time, held more or less regular meetings, but they were pre-eminently for administrative purposes.

Staff meetings, fortunately for all concerned, have now become medical clearing houses, and have been of value in proportion to the interest manifested by the individuals taking part in the work. Cases have become common property and experience consequently widened.

The former methods, whereby, largely through heresy, or on account of unusual interest, only a few came before the staff, have been corrected as far as possible.

There are obvious reasons why the entire staff should share in the results of every examination that has been made by another. Clinical work is better for having been scrutinized and passed upon in a formal way rather than casually brought to notice.

The facts of the history, the mental and physical findings and the patient are presented. All are considered with the view of giving full value to the diagnostic points.

The impressions thus made are less likely to lose their force or fail to be of help in the study and observation of future cases. It is not to be forgotten that purely physical questions should receive their share of attention in relation to psychoses.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

BIBLIOGRAPHY.

1. Benedict and Snell: Archiv für die gesammte Physiologie, 1901, Vol. LXXXVIII, p. 492; 1902, Vol. XC, pp. 33-72.
   Benedict: Amer. Journ. of Physiol., 1904, Vol. XI, pp. 145-169.
2. Oberneir: "Der Hitzschlag" Bonn, 1867.
3. Jürgenson: Der Körperwarme des gesunden Menschen, Leipzig, 1873.
4. Leibermeister and Hoffman: Handbuch d. Path. u. Therap. d. Feibers, Leipzig, 1875.
5. Mosso: Vergleichende Phys. der haussäugethire, 1892.
6. Myers: (Quoted from Benedict.) Yale Med. Journ.
7. Mosso: Die Temperature des Gehirns, Leipzig, 1894.
8. Pembrey: Schäfer's Physiology, London, 1898.
9. Carter: Journ. Nerv. and Ment. Dis., Vol. XVII, p. 785, 1890.
10. Gibson: Journ. Med. Sci., 1905, Vol. CXXIX, p. 1049.
11. Bosanquet: "Annual Heat," Todd's Cyclopædia, Vol. II, p. 659.
12. Riva: Rivista Sperimentale, 1879.
13. Riva: Arch. Soc. Freniatrica, 1883.
14. Croemer: Allg. Zeitschr. für Psych., Vol. XXXVI, Band 2, u. 3 Heft.
15. Wirsch: Centralblatt für Nervenheilkunde, Mar. 1, 1881.
16. Rottenbiller: Centralblatt für Nervenheilkunde, Jan. 1, 1889.
17. Savage: Quoted by Turner, Journ. Ment. Sci., Vol. XXXV, p. 347.
18. Crichton Browne: Quoted by Turner, loc. cit.
19. Peterson and Langdon: Journ. Nerv. and Ment. Dis., Vol. XVIII, p. 750, 1893.
20. Parsons: Journ. Nerv. and Ment. Dis., 1895, Vol. XX, p. 407.
21. Sorokovekoff: Moniteur neurologique, Rus., 1904.
22. Diefendorf: Trans. Amer. Med. Psy. Assoc., 1903.
23. Noyes: Brit. Med. Journ., Vol. II, p. 551.
24. Ott: Jour. Nerv. and Ment. Dis., 1893, Vol. XVIII, p. 774.
    Ott and Scott: Jour. Exp. Med., 1907, Vol. IX, p. 61; Tangl. Pflüger's Arch., 1907.

THE VALUE OF STAFF CONFERENCES IN STATE HOSPITALS.*

By ELBERT M. SOMERS, M. D.,

First Assistant Physician, St. Lawrence State Hospital, Ogdensburg, N. Y.

Staff conferences, essentially medical in character, have been in operation in the various State hospitals of New York State since the year 1905, as required by law.

The introduction and adoption of newer methods of clinical study, shortly before this period, clearly emphasized the need of more uniform and concerted action on the part of hospital staffs with reference to the study of the mental and physical condition of those admitted for care and treatment.

Previously, some of the hospitals had, for some time, held more or less regular meetings, but they were pre-eminently for administrative purposes.

Staff meetings, fortunately for all concerned, have now become medical clearing houses, and have been of value in proportion to the interest manifested by the individuals taking part in the work. Cases have become common property and experience consequently widened.

The former methods, whereby, largely through heresy, or on account of unusual interest, only a few came before the staff, have been corrected as far as possible.

There are obvious reasons why the entire staff should share in the results of every examination that has been made by another. Clinical work is better for having been scrutinized and passed upon in a formal way rather than casually brought to notice.

The facts of the history, the mental and physical findings and the patient are presented. All are considered with the view of giving full value to the diagnostic points.

The impressions thus made are less likely to lose their force or fail to be of help in the study and observation of future cases. It is not to be forgotten that purely physical questions should receive their share of attention in relation to psychoses.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

Criticism under the proper conditions is also a valuable feature in bringing out points of interest that may have been overlooked. The best policy, as to the treatment of a case, is known to all. The impression gained by the patient, that the entire staff is interested in his behalf, aids materially in gaining the good will and confidence of the subject. The various services in institutions become less individualized and apart. They are rather made to contribute whatever may be of medical interest to the general fund, and those in charge draw their experiences from sources broader than formerly, for each member of the staff is continually assigned new cases for study and presentation.

A properly organized staff conference is a step forward in hospital organization, and when it is fully inaugurated there seems to be no good excuse for substituting the time thus spent for other methods of instruction.

Uniform methods of examination with definite times set apart for the mutual consideration, as far as possible, of medical questions, cannot be considered as impracticable, either because too much time may be required or because of fear that other matters of routine will be neglected. The arguments against this procedure can best be made only after a fair trial.

It is probably true that no State hospital staff is so busy that it cannot set apart some portion of time for conference upon strictly medical matters.

The sole object of this paper is to briefly point out some of the elements of value in staff conferences, based upon the assumption that all obstacles for their prosecution have been successfully removed. Therefore, one's description will be largely from personal experience in a hospital where daily conferences are the rule.

In organizing the medical program for such meetings, it should be the duty of the one in charge of the clinical department to assign cases to other members of the staff as soon as they are admitted, and so arrange the work that it be as equally divided as possible. This duty rather naturally falls to the first assistant physician, as he usually has general oversight of the recent admissions and of all clinical matters generally.

In institutions where the annual admissions are not over 400, and the ratio of physicians to the general population is about one

to 170, all the cases can, upon entry, be fully examined and presented a sufficient number of times before their discharge if daily conferences are held. Many of the larger hospitals of this State, however, have admission services so active that even daily conferences could not properly dispose of all the cases. The practical advantages gained by the presentation of as many cases as possible, nevertheless, holds good.

In the selection of cases to be presented, enough material should be provided to completely occupy at least one hour.

It has been found feasible to present all cases under three different conditions. First, within five days after admission. Second, when the case is completely worked up, usually within six weeks. Third, before discharge.

The advantages of the first presentation are of some importance. In New York State there is a statutory regulation that requires the superintendents of hospitals to see all patients within five days after admission, and thereby this requirement is conveniently observed, as the superintendent is naturally the presiding officer during the clinical hour.

At this period, more attention is paid to the points of history than to making a diagnosis, and in addition, to primarily determine whether the patient is a proper subject for detention in the institution. Therefore, the attendant, whose trained duty it was to obtain from the relatives or otherwise as full a history as possible before bringing the patient to the hospital, is also required to be present before the staff in order that the errors of history may be corrected, doubtful points inquired into and fuller descriptions of certain events obtained.[1] In this way, there is often gained additional information of value, the mental level of the relatives and home conditions are better understood, and the history, when recorded, is in better form.

This method of scrutiny of the attendant's report has its obvious advantages and tends to make him more accurate in his inquiries.

Those hospitals, which draw their patients from large rural

[1] The New York State hospitals send, upon notification, trained attendants to the homes of patients, for whom admission has been sought, to convey such patients to the hospital.

sections, depend greatly upon the thoroughness of the attendant's account. The opportunities for personally interviewing the relatives are not as frequent as in compact city districts.

For the sake of a better understanding of the cases, carefully framed letters of inquiry to the relatives or to the family physician of the patient are sometimes necessary, should the attendant for one reason or another fail to gather satisfactory information.

At this presentation, it is not advisable to attempt to examine the patient to any extent, but rather to tactfully explain to him the reasons for his detention and generally to give him the impression, as far as possible, that full consideration will be given his condition. For this manifestation many patients are grateful and others are disposed to be more free in their attitude and conversation when the mental status is seriously sought by the examiner. It rarely does harm.

Incidentally, however, observations at this time are recorded as to the reactions of the patient since entry. The examiner briefly reports whatever points of interest he may have had time to obtain, particularly as to the physical condition, the immediate therapeutic indications and the more important mental features, such as the orientation, evidences of hallucinosis, the character of the productivity or any other features which can be readily demonstrated without taking too much time.

A brief, general survey of the patient, as soon as possible after admission, is of clinical advantage to all and enhances the subsequent impression gained when the case is again presented in a more complete form for diagnosis. Furthermore, this method leaves the records of those who suddenly die, or are removed, in better order. Some things of value may have been noted which would, otherwise, have been missed.

There are, in the course of admissions, some cases which can be properly worked up and disposed of, as far as the physical and mental conclusions are concerned, within the five days. These are principally cases of well-recognized deteriorations, such as the senile and epileptic, and occasionally cases of well-marked general paralysis. But the conclusions, however, should not be accepted until they are apparent to all, and it is not to be forgotten that in these cases interesting conditions subsequently

arise for further study such as neurological or other organic manifestations.

Relatively few patients are in such condition that they cannot be presented before the staff. Those that cannot are so because of some serious physical disorder or, rarely, because of extreme sensitiveness, such a state as we might readily infer in the case of a refined woman, depressed and fatigued upon entry.

The second presentation of the case is made only after the examiner has, as fully as possible, completed his examination. Six weeks are sufficiently long in which to gather and put in typewritten order what is obtained. For the sake of clearness a summary of the findings is submitted rather than the whole case in order to cover in succinct and comprehensive form the principal features of the case. The complete examination can, at any time, be referred to whenever more detail is required concerning some particular point.

It seems not wise to defer longer in hopes of adding anything materially helpful for diagnostic purposes, even if inaccessibility is the stumbling block. There will be a small percentage of cases that cannot be satisfactorily classified even at a more remote period, but enforced postponements for this reason should not deter energetic and painstaking efforts to come to early and fairly sound conclusions in those cases in which the symptoms are demonstrable.

It would add nothing to the value of a case of general paralysis to wait for positive evidences of memory faults before giving the disease a diagnostic name. The exhibition would be of more interest before this stage. The habit of waiting too long before making a diagnosis robs the case of its freshness and interest for others. The salient points become historical rather than clinical and the examiner substitutes convenience for expediency.

It is decidedly more interesting for the staff to witness retardation rather than to take another's word for it. A manic case is more instructive when he can be readily shown to be so. The mood, attitude and manner of the hallucinated person mean more when observed early. A case should be shown when it is worth while if clinical material is to be the means of sharpening our knowledge and rendering us more resourceful.

After the record is presented and the patient properly interviewed and dismissed, discussions are then in order. Herein rests much of the further value of a staff conference. The one who presented the case has been mindful of this when summarizing the record and arranging his diagnostic points. The contentions and arguments incident to such discussions should be pertinent and not allowed to become too miscellaneous or unprofitable.

In the main, the following questions are up for consideration, namely: What is your impression of the case? Do you agree in the conclusions that have been drawn? What features do you see that are unusual or that differ from those manifested in former cases of the same type? Have the therapeutic indications been met? What factors influence the prognosis?

In this way the case is not simply disposed of by merely giving it some diagnostic name. This characterization can be best reserved till the last. The more important consideration is whether we have understood the individual case, and of what use can it be in practical work.

A record of such a presentation should be made by the examiner covering the important features elicited from the patient and the opinions expressed by the various members of the staff. By this means, aid is given in the subsequent observation of the patient in whatever service he may be.

There are occasionally good reasons for again presenting the case to show some unusual mental or physical conditions that may have arisen or because of some obscurity regarding previous symptoms.

The final presentation of a patient is made when his discharge comes up for consideration. This usually takes but little time particularly if the case has recovered. Under any conditions it is well to know as far as possible the exact mental attitude of those who are to leave our charge. A brief review of the main points of the case, together with the course of psychosis, is submitted, and the welfare of the patient, as well as the interests of the community from which he came, are determined by what is found. If the patient is well, some profit accrues from learning under what circumstances and by what method he was able to

readjust himself. What may constitute insight is a feature peculiar to each case.

Lastly, it seems advisable to set apart some portion of an hour during the week for the consideration of any autopsies that may have been held. The findings are considered in relation to the clinical notes. The extent of the continued observation and interest in the cases, which have been more or less disposed of, are hereby measured. The accuracy of the diagnosis of intercurrent physical disorders and of the terminal disease are here put to the test, and oversights become matters for explanation.

It is probable that in the past the necropsy has been put too much apart by itself, and has not been considered in close enough relationship to conditions found existing during life. Autopsy material manifestly belongs to the clinician. It is his case even after death and no autopsy should be conducted unless the examiner is there with the record of the case. Furthermore, the pathologist is thereby better guided in his immediate investigations.

In some such way, as briefly outlined, can the hour given to consultation be made profitable. It is this kind of schooling that will be likely to help the staff to keep pace with the present day requirements of practical psychiatry.

Matters of medical import are brought into better line with something definite in view, namely: Accuracy of observation, correction of wrong impressions and construction of permanent records, which contain more complete and orderly data for future help. In addition good opportunities exist for subjecting to practical test whatever may be found in general literature and mere book knowledge takes its proper level.

Daily conferences give the superintendent a greater personal knowledge of the general medical activity of his staff and he is in a better position to measure the value of each individual.

REVIEW OF INFECTIVE-EXHAUSTIVE PSYCHOSES WITH SPECIAL REFERENCE TO SUBDIVISION AND PROGNOSIS.*

By SAMUEL W. HAMILTON, M. D.,[1]

Second Assistant Physician, Utica State Hospital, Utica, N. Y.

In the New York State Hospital classification as projected by Dr. Adolf Meyer one of the divisions is called the infective-exhaustive group. It includes "autotoxic or infective or exhaustive psychoses not included in the group with a nervous disease or nervous complex or tangible brain disease; subdivided into (a) thyrogenous disorders, (b) uremic, eclamptic and demonstrated gastro-intestinal disorders, (c) febrile and postfebrile deliria, (d) exhaustive deliria and kindred psychoses." An allied group includes "those to all intents and purposes the same, but in which we cannot demonstrate such an etiology." Such cases have been in the past grouped in various ways by different writers. Let us review a few.

Ziehen describes acute hallucinatory paranoia which arises from exhaustion, intoxication, infection, trauma, hysteria and epilepsy, polyneuritis, puberty, climacteric, senility or the puerperium. Its diagnosis depends on proof of primary hallucinations which prevail throughout. Out of these develop delusions. Affect and association are only secondarily disturbed. With its varieties and subdivisions this would seem to include so many cases as to be of little help to us in any direction. For example there is the greatest possible range of prognosis. Moreover we find in cases otherwise similar that hallucinations were not prominent, and the existence of these can be determined only on recovery.

Krafft-Ebing object that this is not a paranoia and does not pass into a paranoia. He calls it primary hallucinatory insanity.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

[1] To Dr. Wm. Mabon, superintendent and medical director of Manhattan State Hospital, I am indebted for permission to use the hospital records; and to Dr. George H. Kirby, director of clinical psychiatry, for the impetus to this study and very many suggestions as to the form and content of this paper.

It is to be diagnosed from mania and melancholia because the behavior is purely reactive. Constant symptoms are poor sleep, nervous excitement and confusion of ideas.

Ziehen presents a large group of conditions of clouded consciousness. These are alike in that the beginning and ending are very abrupt, course very short, amnesia follows; hallucinations and delusions may be absent, but the orientation and connection of ideas are disturbed. The principal causes are sleep, epilepsy, hysteria, alcohol and other toxic drugs, congestion or spasm of the arteries, migraine, neuralgia, strong affect, trauma. We need not discuss these varieties, which indeed from their symptomatology could hardly be differentiated. Accompanying or symptomatic deliria are distinguished from the preceding groups in that the course is closely related to the infection.

Wernicke describes in his own peculiar terminology confused mania or agitated confusion. There are psychomotor or psychosensory disorders and dominant symptoms of irritation. An asthenic confusion also with allopsychic disorientation is met after other exhaustive psychoses.

Kraepelin attempts to distinguish infection from exhaustion psychoses. The first include fever delirium, infection delirium (such as arises during the incubation period of some severe infection) and post-infection mental debility appearing in convalescence on the ground of a fever or infection delirium. Exhaustion psychoses are collapse delirium at the crisis of some fever, acute confusion or amentia, and chronic acquired nervous exhaustion, which differs from other nervous exhaustions merely in that its starting point is some severe illness or strain. Thus his divisions are determined partly by the stage of the illness at which the disturbance develops, partly by the form of the psychosis.

Observe Kraepelin's types briefly with reference to experience at Manhattan State Hospital. Few fever deliria reach us, for it is the policy of Bellevue Hospital, whence most of our patients come, to transfer as few fever cases as possible. Infectious deliria, since they usually precede the outbreak of some severe illness, are sifted out by the general hospitals of New York City. Collapse delirium occurs in a state of such weakness that transfer to a state hospital would be unlikely. Chronic acquired nervous exhaustion is not certainly the result of that alone which appears as

the upsetting factor, and is generally not grouped by us with the infective-exhaustive cases. The bulk of those left are post-infection mental debilities or similar states apparently based on poor health and in a few cases on prolonged worry. Amentia is the only remaining type, and I do not find it frequent.

Kraepelin divides these debilities into four degrees: The first shows mental and emotional weakness; he is dull, tired, unable to collect his thoughts, may see vivid pictures when his eyes are closed. There are premonitions of death, suspicions leading to violence, attempts at suicide. The second stage shows prominent hallucinations, fantastic delusions, anxious excitement, profound obscuration, disconnected talk. There are dead people behind the bed, walls and furniture move, devil or Virgin appears, he does not know whether he is in heaven or hell, is given horsemeat, and so on. Recovery is slow with decreased efficiency and weak memory. The third subdivision passes rapidly into stupor, and patient later continues stupid or tearful with deficient memory and judgment. In the fourth degree occurs vivid delirious excitement with marked flight, delusions of grandeur, loss of orientation, great distractibility, voices, angels at the ceiling and such like. Misidentifications are frequent. Particularly with the third group (he says) the patient may remain mentally and emotionally incapable, apathetic, undecided, but apart from these defects he assumes a favorable outcome by assuming that any other is due to a different process. "In a few cases . . . . develop . . . . mental diseases which might also occur under other conditions—manic-depressive insanity, dementia præcox, general paralysis. In these cases the fever is not the cause but the occasion."

I wish to review with you a number of cases (22) observed at Manhattan State Hospital to see if they throw any light on the composition and outcome of the infective-exhaustive group. It must be admitted that patients with adequate etiology, symptoms apparently sufficient for classing with this group, notwithstanding show deterioration indistinguishable from dementia præcox, and a few such cases are considered in this list. Cases ending in death are with one exception excluded, since they do not illuminate the issues. We will divide them so far as possible into Kraepelin's degrees of severity.

A comparatively mild case was K. L. During typhoid fever

she jumped out of bed, demanded a priest, said she was going to die. She would not eat for a time, complained that her arms were heavy, misidentified, replied only when urged, thought people were calling her. She grasped mental tests slowly and avoided mental effort, refusing to count change with the words " It puzzles me." It was four months before her normal state returned fully though she left the hospital earlier. F. P. during puerperal fever became " delirious," then depressed, and had to be tube-fed. She lost track of time and movement from one hospital to another, saw faces and pictures on the wall, heard voices at night. With us she was afraid the patients would hurt her, thought one was mocking her by belching, said the medicine tasted like paint, heard voices of enemies. Soon she cleared up but had little idea of time duration during the hazy period. J. G. after uterine hemorrhage thought someone wanted to kill her, that they tried to get into her room at night. She talked freely but incoherently, was worried and restless, complained that the food was not what she wanted, that she was lonesome. Recovery in nine months. Another typhoid case with different outcome was P. M. During convalescence he became moody and once was excited, throwing his chair about. On going home from the general hospital where he had been sick he sang, shouted and thought he was driving. Gradually he reached a state where he complained of books being put in his head, that his thoughts were read, that machines were used on him. He thought Central Islip was Blackwell's Island. He is discontented, idle, surly, irritable. These four cases would fall in Kraepelin's first division.

The second degree with prominent hallucinations, fantastic delusions, etc., were more numerous. C. T., after a period of poor health, grew confused and could hardly find her way around the streets of a city she had formerly known well and was now visiting. She grew uneasy and agitated, smelled peculiar odors and saw " such horrible people " staring. She misidentified strangers as members of her family, thought the blowing off of a gas retort was a deadly explosion and shouted for the police between mouthfuls of her supper. Her husband's voice came from outside the building. Later she tried determinedly to kill herself. Gradually she became clear. A. F., in the eighth month of lactation, while in poor general health, developed peculiar bodily sensations which

she thought due to poison. Then she talked of Black Hand persecution, heard Italians at the foot of the stairs and negroes on the roof. Once she did not recognize her husband, saying, "This is the man that killed my baby." Symbols of the Black Hand appeared everywhere; she was annoyed by hallucinations of sight and hearing. A newspaper portrait she thought hers and two physicians her brothers. She ran about the place half clad, thought it was the House of the Good Shepherd. For a time she was cheerful, flippant, sometimes resistive. Then she recovered. A. S.'s trouble started during the fever of gastro-enteritis. In a general hospital she misinterpreted all sorts of sounds and tried to choke the physician who, by giving her mercury, had caused all her troubles. Her mood, arising out of grandiose delusions, was cheerful; nevertheless she was suspicious. She never gained complete insight, but has done well outside the hospital a long time. H. H. was worried over sexual irregularities and working unnecessarily hard during the weeks following labor. She saw ghosts, cats and dogs. Misidentifications, perplexity, her husband's voice from the floor, disorientation are noted. Improvement came slowly and judgment lagged behind other faculties. B. P. suffered general ill health and was grieved by the death of her children. During another pregnancy she heard fire engines, people talking outside and shots fired by people who were coming to kill her husband. Her speech became incoherent, but she was evidently intensely worried. She refused food, got extremely suspicious and apprehensive. Remarks by other patients she thought were aimed at her; people said she had coons for brothers. Gradually she gained strength and courage. L. N., during convalescence from erysipelas, thought people would kill her and believed herself poisoned. Orientation was hazy and she was suspicious and fearful. At time of discharge she would not yet speak unreservedly of her experiences but did mention seeing and hearing things. F. K. had poor health and nephritis. She complained that she was not given food, predicted that the doctor's medicine would kill her, told in what clothes she was to be laid out. Violence, an attempt at suicide and incoherent speech followed. Her most prominent hallucinations were tactile. She thought the place a telegraph office and could tell only the month when a calendar was before her. Other patients she thought men and all talked

ragtime. Improvement came slowly, and as in other cases amnesia remained.

Two similar cases failed to recover. J. H. had pneumonia, got out of bed, was unreasonable and violent, called out the window to friends both dead and alive. She was semistuporous for a while, heard funny voices through the wall. Then she admitted having been mixed up in the head, but was averse, sometimes resistive, always silly and indifferent. So she remains. N. H. had hallucinations and did very peculiar things a half year before commitment, but was employed at her occupation of nurse, when an infected finger led to a further mental upset. She was depressed, confused, apprehensive, talking of visits at night from her dead mother, the Virgin passing hands over her face, prospective death by murder. Her delusions grew more absurd and extensive and are now incoherent. She talks incessantly without goal. On account of the development during septicemia of the trouble that led to her commitment her psychosis was at first thought to belong to this group.

The next is a transition case to the third—the stuporous—variety. A. G. was nursing her three-months-old infant in spite of deteriorating health when she began to speak of extensive purchases and to say she felt as if going crazy. She thought everybody was talking about her and that the landlord had an ambulance waiting for her. Vivid hallucinations and fear of death succeeded; then for ten days she did not speak. She passed through several fantastic experiences, being put through a boiler and down a tunnel. When other patients spoke on indifferent topics they seemed to be defaming her. For a time her orientation was obscured. D. K., during lactation and hard work dreamed of ghosts and coffins, saw men walking on the ceiling and her children jumping out the window. Voices threatened to cut her head off and bury her. Everything was "doubled." Two months she was mute. A. B. had longstanding uterine trouble. She heard neighbors planning to rob her flat and appealed to the police for protection, but grew rapidly confused and incoherent, and suspicious and fearful by reason of her hallucinations. Though willing to talk she could not control the train of thought. For three weeks she did not speak and for some time longer showed variable responsiveness. C. B. had pericarditis and mistook identities,

complained that her father was talking about her, shouted incoherently at times, but soon sank into stupor, retaining saliva and urine, resisting passively and requiring to be fed by spoon. When in other regards improving she still thought she was to be killed. F. H., while in hospital with superficial infection after hysterectomy, became confused in speech and expected to be killed. She screamed with terror, resisted attention violently, repeated phrases but did not answer questions. More than a year has elapsed and she is still mute, inactive, resistive.

Fourth stage—violent excitement: L. M. had a post-partum hemorrhage. She was urged to get up and bathe on the seventh day, and attacked her mother-in-law with an axe. She passed quickly through a wild, apprehensive excitement associated with evanescent hallucinosis. She was kept in the hospital some time after apparent recovery, but on the fourth day after discharge got confused and noisy while out walking, and after a week of violent excitement died suddenly. S. S., after suffering some months from an exhausting vaginal discharge, spoke incoherently and with many repetitions, rushed about shrieking wildly, reacting to fearful auditory hallucinations. The degree of excitement fluctuated but was always notable. Gradually she became quiet and gained insight.

This brings us to a patient who exhibited flight at one time, depression later. M. D. during puerperium said she was the Virgin, abused everybody, did not sleep, refused food. She showed tremendous excitement with fear. A few fair samples of flight were recorded. After three days out of bed she entered another excitement in which she talked with her father under the bed, misidentified, had many rapidly changing delusional ideas. On emerging from this she was unduly sober and felt unable to do things.

A possible amentia in Kraepelin's sense, but without adequate physical basis and with only moderately upsetting mental factors, was the psychosis of L. B., who thought that everybody was dead, that they wanted to kill her. She seemed dazed, commented on all that happened, but evidently could not grasp the situation. Hallucinations were present, orientation much disturbed, and her ideas changed rapidly. Death in various forms was threatened, and she thought that injury had already been done to her children.

Clearly the subdivisions overlap. Let me instance a case that fits in nowhere. M. W. went home from a general hospital after typhoid, apparently well. A week later she began to do peculiar things, such as to demand, "Look at me!" without giving a reason. She would not believe that her brother had left the house and searched for him in drawers and under the bed. Solemnly she confessed various imaginary wrongdoings. Four months she was in another institution; we do not know what happened there, but on transfer she was much befogged, did not recognize her brother and on the trip to New York repeated monotonously, "Open the door!" For hours she would chant a few sentences monotonously; at times the productions could be influenced by what was said to her. In a few days she became accessible and showed defective orientation and amnesia for some four months. She was removed from the hospital without insight and in an irritable frame, but was reported later as having recovered.

A difficult question is the separation of deteriorating cases from recoverable. As you have observed, several of these patients did poorly; some of these had a mild delirium, some a moderate, some a severe. It would have been easy to add others in which the diagnosis infective-exhaustive psychosis was made at one time or another, but without what now appears satisfactory reason. Among them typhoid fever, pneumonia, postoperative infection appear as etiology, but this does not help us; in the same series are recovered cases with typhoid and puerperal infections. Nor can we say as yet that the previous mental makeup determines: P. M. was headstrong, F. H. secretive; but among the recovered cases F. K. was unreasonable to the point of violence, A. S. stubborn and not given to forming friendships. Perhaps if the study of the mental makeup were carried further it might yield the same help as in other fields. We can at present say only that delirium is so natural and fundamental a reaction to severe disturbance of the human economy that it is quite unsafe to attempt prognosis or more than tentative diagnosis till the affair has had time for the causal lesion to be repaired and for incidental features to disappear. Let us accumulate careful observations; the last word on delirium is not spoken. For subdivisions of the group some simple scheme of degree of severity seems as useful as anything more elaborate and etiological when admissions are sifted as are those in New York city.

STUDIES IN HEREDITY WITH EXAMPLES.*

By WILLIAM C. SANDY, M. D.,
Assistant Physician, State Hospital, Trenton, N. J.

Heredity has long been recognized as an important etiological factor in mental disease. The term, however, has been loosely applied and indefinitely stated. It is usually considered sufficient to state the percentage in which hereditary influences appear in the different psychoses without any attempt to specify the character of these defects. In the present paper an attempt is made to determine more definitely what psychoses or peculiarities are found in the families of a series of cases in the institution with which the writer is connected.

At the very onset it is well to consider the difficulties encountered in such a study. The hospital records constitute one of the important sources of information. One who has had any experience with old asylum histories, however, need not be told that these are often meagre and valueless. Moreover, when an honest effort is made to obtain useful data the informant is frequently unreliable. He may be ignorant or may purposely distort or conceal the real facts. Even if the informant be reliable, yet there is no standard in measuring mental conditions. One must consider also the education of the individual, his environment, opportunities, and so forth, before an equitable judgment of his mentality can be made. Again it is difficult to decide the relation of the peculiarity in ancestors to the psychosis in the case at hand. The fact that a history of insanity or peculiarity may be obtained, after careful search, in the families of many normal individuals must be borne in mind, and the danger of a *post hoc propter hoc* argument avoided.

The biologists are accomplishing much, which will doubtless help to solve the problems of the hereditary etiology of mental disease. A number of analogies may be found between the unit characters of the anatomical and those of the mental make-up. The time may come when, as Prof. Davenport has said, if the

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

characters of the parents are known, it will be possible to predict the kind of children.

In this regard it is important to make careful and minute studies of individuals in families in respect to certain traits or unit characters and their transmission, disappearance, or change in successive generations. In order to eliminate, so far as possible, the element of difference in education, environment, and opportunities, Strohmayer suggests that the families studied should be from a small little-fluctuating community.

The problem then is extremely complex. The present examination scheme, as usually followed, is not sufficient to collect really valuable data to establish the influence of heredity. To one in search of facts, even the so-called well-worked-up records are exceedingly disappointing.

Besides studying the psychoses in relatives and endeavoring to show in this way a real hereditary influence, it is also well to consider to what extent acquired conditions may affect the descendants. Although some writers dispute the possibility of the transmission of acquired characters, yet it must be acknowledged that such habits as alcoholism in the ascendants leave their mark upon the inheritors. Alcoholism in the parents, especially at the time of conception, has been shown, by more than one authority, to result in the propagation of defectives.

In the examination of the present series of three hundred and eighty-six cases, two hundred and twenty-nine men and one hundred and fifty-seven women, special attention has been paid to the psychoses, peculiarities or toxic habits in paternal and maternal grandparents, uncles and aunts, parents, brothers and sisters. Many of the statistical findings are not greatly different from those of numerous writers on the subject. Of the three hundred and eighty-six cases, one hundred were in the manic depressive group, sixty-eight dementia præcox, fifty-two alcoholic insanity, eighteen general paresis, twenty-three epileptics, six narcotic inebriety, nine imbeciles and one hundred and ten cases in which there was found to be apparently very little hereditary influence and which are grouped together. Ninety-five cases, or a percentage of 24.61 of the whole number, showed undoubted heredity, such as psychoses or eccentricity in ancestors. If those

cases were added which gave a family history of alcoholism only the percentage becomes 35.

STATISTICS OF HEREDITY.

Form of Insanity.	No. of cases.			No. showing heredity.			Heredity percentage.		
	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.
Manic Depressive	47	53	100	19	10	29	40+	18.8	29
Dementia Præcox	43	25	68	13	4	17	30+	16	25
Alcoholic Insanity	46	6	52	15	0	15	32.6	0	28.8
General Paresis	13	5	18	7	0	7	53.8	0	38.8
Epilepsy	17	6	23	7	1	8	41+	16.6	34.7
Narcotic Inebriety	6	0	6	4	0	4	66.6	0	66.6
Imbecility	6	3	9	4	0	4	66.6	0	44.4
Other Psychoses	51	59	110	3	8	11	5.8	13.5	10
Grand Totals	229	157	386	72	23	95	31+	14.6	24.6

In the manic depressive group, one hundred cases, forty-seven men and fifty-three women, there was an hereditary taint of 40.42 per cent in the former and 18.86 per cent in the latter, or a total of 29 per cent. While most observers agree that the male cases show a greater percentage of heredity than the female, yet the difference is not usually so marked as in the present series.

As has already been said, it is impossible always to state even the probable diagnosis in the cases of insanity in the relatives. A number, however, show evidences of manic depressive insanity, both by individual symptoms, namely depression or excitement with recovery, and by recurrence of the attack. Five were suicides, very likely constituting depression. One case had a maternal grandmother who had a psychosis following childbirth from which she died. His mother was twice admitted with maniacal symptoms, recovering completely from the first attack and staying out of the hospital for eighteen years. Her second hospital residence has been of twenty years' duration, and now she seems to be a senile dement. His sister is also of the manic depressive group. There were seven other probable cases of manic depressive in the relatives, eight of apoplexy or paralysis, a few nervous, neurotic or deficient, two epileptics, several senile dements, a number classed as simply "insane." It is interesting and important to record a well-marked case of dementia præcox,

brother of a female manic depressive—the præcox exhibits the characteristic apathy, dementia, mutism and resistance.

In eight male cases, or 17 per cent, a history of alcoholism with no psychosis was found. If this is added to the percentage already obtained, a total of 57.44 per cent among the males results and a grand total of 37 per cent. Comparing these figures with those usually given the difference is marked. Paton gives the percentage as between 80 and 90; Diefendorf, 70 to 80.

Of the sixty-eight dementia præcox cases, seventeen, or 25 per cent, showed evidence of heredity. There were forty-three men with 30.23 per cent and twenty-five women with 16 per cent hereditary influences. Twelve out of the seventeen cases showing heredity have relatives who are designated as neurotic, peculiar, queer, defective, or as having the same psychosis. In seven there was no psychosis, only the peculiarity or eccentricity. In one case, there was a history of brain abscess in paternal grandfather, paralysis in paternal grandmother, excitement, with recovery, in maternal grandmother and paternal uncle, the latter two possibly manic depressive cases. Some doubt was cast upon the diagnosis of dementia præcox, however, by the complete disappearance in a few days after admission of all symptoms. Some favored a diagnosis of hysterical insanity, others a remission in a præcox. In two instances it was impossible to determine the form of insanity in the relative, one being the father, the other the maternal uncle of the patient. A maternal uncle of one female patient was twice an inmate of this hospital. The records state he was always considered queer. He remained only five months the first time, his age being forty-five. The second admission occurred six years later and from that time until his death, twenty-four years afterwards, he was confined in the hospital. His death was supposed to be from cerebral hemorrhage (post-mortem examination not being allowed). The records state he showed gradual deterioration with dementia towards the end.

There seems, therefore, to be some basis for the statement that in dementia præcox the hereditary influence in the ancestors is frequently a peculiarity, eccentricity or defect causing the persons to be designated as neurotic or queer. Also that the same psychosis is apt to appear in the relatives.

As in the manic depressive group, so also in dementia præcox,

alcoholism in the family history seems to be quite common. There were eight cases which showed alcoholism only in relatives. If these be added to the cases showing insanity or peculiarity the total percentage of heredity is 36.76. There were four cases giving a family history of insanity which also had alcoholic relatives. Most of the alcoholic taint came through the father.

All those cases in which the psychosis or trouble was some form of alcoholism have been placed in the alcoholic insanity group. Thus, chronic alcoholism, alcoholic hallucinosis, alcoholic paranoia and delirium tremens are grouped together.

There were fifty-two cases, forty-six men, six women. Fifteen of the men, or 32.6 per cent, showed some form of insanity or peculiarity in relatives. None of the women gave any evidence of heredity. There were among the relatives three cases of senile dementia, three of paralysis or apoplexy, two of epilepsy or convulsions, five nervous, hysterical or eccentric, five depressions, one being a suicide. The above calculations leave out of consideration any alcoholism in relatives. Nineteen showed alcoholism only in ancestors, eight alcoholism and insanity. The percentage of alcoholism above added to that of psychosis or peculiarity brings the average up to 73.9 per cent.

There seems to be nothing striking or characteristic about the kind of psychoses in relatives of cases of alcoholic insanity. The percentage of alcoholism in ancestors, however, is large and should serve to emphasize the importance of this etiological factor in mental disease.

General paralysis is commonly thought of as a disease in which heredity is of little importance as an etiological factor. Wiglesworth concluded that paresis has less hereditary causation than other forms of insanity. Diefendorf, on the other hand, gives an heredity of 50 per cent. Paton states that the consensus of opinion "favors the view that the family history indicates the existence of nervous or mental trouble in the ancestors in at least 45 per cent of all cases."

In this series there were only eighteen cases of paresis, thirteen men, five women. The latter showed no hereditary influences—the former, 53.84 per cent. If the female cases are included the total percentage becomes 38.88. The specific instances of insanity or peculiarity in the relatives, however, were rather scattered and perhaps difficult to connect with the paretic cases. For

instance there were found alcoholism in the father and paralysis in the maternal aunt in one case, queerness in father and depression in brother in another, senile dementia in maternal uncle in still another, alcoholism in father and brother, insanity in maternal grandfather, insanity in brother and lastly apoplexy in father. When one takes into consideration the mass of evidence supporting the syphilitic etiology of paresis, such hereditary influences as have been found in these cases probably play a minor part.

The epileptic group, twenty-three cases, seventeen men and six women, have eight cases (seven men, one woman) showing hereditary influences in the men, 41.17 per cent; in the women, 16.66 per cent, a total of 34.78 per cent.

Among the psychoses or peculiarities of the relatives there were five insane, one maternal grandmother, two maternal grandfathers, two mothers, form of psychosis unknown, one insane brother, a well-marked catatonic præcox, three cases of paralysis—two in fathers and one in mother—one alcoholic dementia in father and one extremely irritable, alcoholic mother. In one case there was a sister diagnosed as arrested development. In two others, there was alcohol in father and mother alone, bringing the total percentage of heredity up to 43.47. Six cases showed some alcoholic relatives, four of these being accompanied by a history of some psychosis or peculiarity.

Six cases of narcotic inebriety, morphinism, all males, gave a high percentage of heredity. Every one of these showed some peculiarity in relatives, either psychoses, alcohol or drug habit. Four, or 66.66 per cent, had relatives with some form of mental disease. One had a maternal aunt and a sister in the institution, both recovering, the form of psychosis in the aunt unknown, that of sister being manic depressive—depressed phase. The father of another was a hemiplegiac and the maternal aunt insane, form unknown. In a third case there was apoplexy in the father, the mother nervous, maternal uncle a suicide, a brother paralyzed and a morphine habitué, the sister nervous. The fourth case showed apoplexy in maternal grandfather and paternal uncle, the father and mother being nervous. Four cases showed also an alcoholic and morphine history in relatives, that being the sole peculiarity in two cases.

The nine imbeciles likewise gave a high percentage of hereditary taint. There were six men and three women, the latter giving

negative family histories. Four of the men, or 66.66 per cent, showed actual psychosis or peculiarity. In one the father was markedly eccentric. A second, the maternal grandfather was insane, form unknown. A third showed senile dementia in father, neurotic mother. A fourth, a half-brother was a deaf mute and a sister neurotic. Besides these, two more gave evidence of alcoholism, one in both parents, the other in father only. The total percentage showing psychoses or peculiarities was, therefore, 44.44 per cent, or if those are included which gave an alcoholic family history only, 66.66 per cent.

Under the heading, "Other Psychoses," a number of conditions are grouped, which have not given such a large percentage of heredity, or of which there are only a few cases. These are paranoid condition, senile dementia, arterio-sclerosis, constitutional inferiority, hysterical insanity and unclassified—a total of one hundred and ten cases, fifty-one men and fifty-nine women. Three of the men, cases of constitutional inferiority, showed psychoses or peculiarities in relatives. One, diagnosed as hereditary cerebellar ataxia, gave evidence of the same disease in mother and brother. A second had an alcoholic paternal grandfather and father, and a maternal aunt who had a psychosis from which she recovered. A third had a neurotic father and paternal uncle, and an insane maternal uncle, form unknown.

Eight of the women showed heredity. One, a case of delirium, had a sister an apoplectic. A case of hysterical insanity had a mother insane, the psychosis being a recurrent type. The father and sister of a constitutional inferiority with hysterical complexes were insane, form unknown. A senile dement had an insane sister, form unknown. An arterio-sclerotic, with hemiplegia, showed mother and paternal uncle insane, form unknown. A case of involutional insanity had a brother and a sister insane, with recovery. Two paranoid conditions showed heredity, one had two præcox brothers and a feeble-minded brother, the other a sister having a recurrent form of insanity.

Returning to a general consideration of the findings, one is led to conclude from the present series of cases that the percentage of hereditary influences is much lower than that usually given. The attention has already been directed to the much lower rate found, especially in manic depressive and dementia præcox. A certain proportion of the discrepancy can doubtless be accounted

MANIC-DEPRESSIVE GROUP.

Name.	P. Gr-Fa.	P. Gr-Mo.	M. Gr-Fa.	M. Gr-Mo.	Father.
Male—					
A. C.				In. fol. c. b.	
A. J.				Stroke.	
G. S.					
C. V. C.			Apoplexy.		Al. Paral.
P. P. W.					
E. B.					
C. C. S.					Dep. Sui.
L. J. D.					Paralysis.
F. G.					
M. H.	Insane.				
H. H.					Alcoholic.
F. B.			Ins. Sui.		Alcoholic.
E. B. L.					Insane.
N. M.					
R. P.					Neurotic.
T. C.					
T. P.					Insane.
H. M.					M. D. (?)
W. E. C.					
Female—					
M. S.		Insane.			
I. S.					Rec. Insane.
A. H.	S. D.	Insane.			Alcoholic.
A. L.					Alcoholic.
M. A.					Insane.
A. N.				Insane.	
J. M.	S. D.	Relig. Ins.			Alcoholic.
K. P.					
K. K.					
L. V.					
Alcoholism only (all male cases):					
J. A.					Alcoholic.
J. M.					Mod. Alcol.
H. R.					Alcoholic.
J. D.					Alcoholic.
J. R.					Alcoholic.
G. S.					Alcoholic.
H. B.					Alcoholic.
A. K.					Alcoholic.

DEMENTIA PRÆCOX.

Male—					
C. J.	Peculiar.				M. D. D. P. (?)
M. G.					Alcoholic.
G. E.					
P. H.	Br. Abscess.	Paralysis.		Insane.	
P. O.					
N. E.					Peculiar.
J. O.					Alcoholic.
M. K.		Stroke.		Insane.	
C. J.	Alcoholic.				Alcoholic.
J. R.					Al. Hemipl'g.
C. B.					
J. DeF.					
F. S.					Defective.
A. W.	Insane.				Eccentric.
Female—					
J. W.					Insane.
A. B.			Insane.		
A. LaR.					
E. A.					D. P.
Alcoholism in relatives only (all male cases):					
H. B.					Alcoholic.
J. D.					Alcoholic.
A. S.					Alcoholic.
J. K.					Alcoholic.
F. J.					Alcoholic.
S. R.					Alcoholic.
U. P.					Alcoholic.
I. F.					Alcoholic.

ALCOHOLIC INSANITY.

G. G.					
J. MacM.					Alcoholic.
M. L.					
F. R.					
P. D.					
A. T.					
W. F.	Alcoholic.		Alcoholic.		Alcoholic.
S. G.	Demented.				Alcoholic.
C. V.					Melancholi.
C. D.					

MANIC-DEPRESSIVE GROUP.

Mother.	Pat. Uncle.	Pat. Aunt.	Mat. Uncle.	Mat. Aunt.	Brother.	Sister.
M. D. S. D.						M. D.
Dep. Sui.					2 Alcoholic.	
Apoplexy.			M. D.			
Nervous.		Insane.				
	2 Paral.					
					M. D.	
	2 Insane.					
	Br. Ab. Sui.					
			Insane.			
					Insane.	
Neurotic.					Peculiar.	Peculiar.
S. D.					Defective.	
		Insane.				
					Ins. Fb. Md.	Ins. Sui.
Insane.					Rec. Ins.	
						Insane.
Paral. Nerv.						Rec. Ins.
Deficient.					Epilepsy.	Nervous.
				Insane.		
				Insane.	D. P.	

DEMENTIA PRÆCOX.

			Insane.			
Ins. (?) Pec.						
	Insane.					
Ins. Pec.						
Peculiar.						
Peculiar.						Queer.
Neurotic.						
Nervous.	Alcoholic.					
					D. P.	
					D. P.	
					Defective.	Defective.
	Insane.					
		Defective.	Defective.			
				Insane.		
	Alcoholic.				2 Alcoholic.	
Alcoholic.						
					Alcoholic.	

ALCOHOLIC INSANITY.

					Insane.	
S. D.						
	Insane.		Eccentric.			
Paralysis.						
			Epileptic.		Alcoholic.	
			Convulsions.			
Nervous.			Sui. Ins.	Insane.		
Eccentric.			Alcoholic.			

ALCOHOLIC INSANITY—Continued.

Name.	P. Gr-Fa.	P. Gr-Mo.	M. Gr-Fa.	M. Gr-Mo.	Father.
J. H.					Al. Paral.
A. C.		Apoplexy.	Sen. Dem.	Nervous.	
J. H.					Alcoholic.
L. C.					
Alcoholism only:					
T. B.					Alcoholic.
J. MacM.					Alcoholic.
T. R.					Alcoholic.
C. S.					Alcoholic.
L. U.					Alcoholic.
F. C.	Alcoholic.				
T. MacC.					
I. H.					Alcoholic.
J. K.					Alcoholic.
F. L.					Alcoholic.
T. A.					Alcoholic.
N. G.					Alcoholic.
T. P.					Alcoholic.
W. F.					Alcoholic.
T. L. C.					Alcoholic.
T. B.					Alcoholic.
S. P.					Alcoholic.
T. K.					
N. N. H.					Alcoholic.
GENERAL PARESIS.					
J. C.					Alcoholic.
H. S.					Queer.
H. M.					
J. D.			Insane.		
T. C.					
E. B.					Apoplexy.
T. P.					Alcoholic.
EPILEPSY.					
W. K.				Insane.	
S. B.					Al. Demen.
D. W.					
C. H.					Paralysis.
D. C.					Alcoholic.
R. E.			Insane.		
A. L.			Insane.		
C. W.					Alcoholic.
W. C.					
A. Van c. F.					Paralysis.
NARCOTIC INEBRIETY.					
W. B.					Mod. Alc.
H. C.					Hemiples.
U. S.					Al. Apoplexy
G. S.			Apoplexy.		Nervous.
A. T.					Alcoholic.
J. L.					
IMBECILITY.					
F. S.					Eccentric.
W. B.			Insane.		
F. P.					S. D.
G. H.					Alcoholic.
C. H.					Alcoholic.
OTHER PSYCHOSES.					
Constitutional Inferiority:					
F. M.					
W. C.					Neurotic.
A. M.	Alcoholic.				Alcoholic.
Paranoid Condition:					
M. L.					
R. N.					
Involutional Psychosis.	*Arterio-sclerosis:*				
N. W.					
Arterio-sclerosis. Hemiplegia:					
E. A.					
Senile Dementia:					
M. C.					
Hysteria:					
A. S.					Insane.
M. P.					
Delirium:					
M. Z.					

STATISTICS—Continued.

ALCOHOLIC INSANITY—Continued.

Mother.	Pat. Uncle.	Pat. Aunt.	Mat. Uncle.	Mat. Aunt.	Brother.	Sister.
						Insane.
Hysteria.						
						Insane.
Man. Dep.					Ins. Dep.	Ins. Dep.
	Alcoholic.				Alcoholic.	
Same Alc.						
	Alcoholic.					
	Alcoholic.				Alcoholic.	
	Alcoholic.					
	Alcoholic.					
	Alcoholic.				Alcoholic.	
	Alc. Several.				Alc. Sev.	
					Mod. Alc.	
			Mod. Alc.			
					Alcoholic.	
					3 Alcoholic.	
Alcoholic.						
					2 Alcoholic.	

GENERAL PARESIS.

				Paralysis.		
					Ins. Dep.	
			Sen. Dem.			
					Insane.	
					Alcoholic.	

EPILEPSY.

Insane.					D. P.	
Al. Irrit.						
Hemipleg.						
Insane.						
	Alcoholic.	Alcoholic.	Alcoholic.	Alcoholic.	Alcoholic.	
Alcoholic.						
						Arr. Dev.

NARCOTIC INEBRIETY.

	Alcoholic.	Ins. Recov.				M. D.
				Insane.		
Nervous.			Suicide.		Par. Morph.	Nervous.
Nervous.	Apoplexy.				Opium.	
						Al. Morph.

IMBECILITY.

Neurotic.					Deaf Mute.	Neurotic.
Alcoholic.						

OTHER PSYCHOSES.

Same.					Same.	
	Peculiar.		Insane.			
				Ins. Recov.		
					2 D. Pr. I.	F. Mnd.
						Re. Ins.
					Insane.	Ins. Recov.
Insane.	Insane.					Insane.
						Insane.
Ins. Recov.						
						Apoplexy.

for by the failure to discover the existing hereditary taint. The difference is so great, however, as to make one think that perhaps too much stress has been put upon heredity.

Besides manic depressive and dementia præcox, several other groups showed considerable hereditary influence. In alcoholic insanity the large percentage of alcoholic family history is the striking feature.

General paresis apparently showed a large percentage, but the influence of heredity is probably not as great as the figures might imply.

Epilepsy gave a heredity of both psychoses and alcoholism.

Psychoses, peculiarities and habits among relatives were common in narcotic inebriety.

Imbecility likewise showed considerable hereditary influence.

The frequency of alcoholic family history in certain psychoses is quite marked. As might be expected, the largest percentage of alcoholic heredity was shown in alcoholic insanity. In the manic depressive group and dementia præcox also, alcoholism in the relatives was far from being unusual.

While it may not be possible from the present evidence to conclude with absolute certainty as to the kind of psychosis in the relatives of manic depressive and dementia præcox cases, yet a few general statements are possible.

The prevalence of psychoses in the manic depressive group as compared with the percentage of the peculiar or eccentric type in dementia præcox is noticeable. In the manic depressive cases, although there is frequently doubt as to the character of the psychosis, the evidence seems strong as to the existence of some kind of a psychosis. On the other hand, in dementia præcox the relative is often described as queer, eccentric, or by some similar term.

The evidence seems to be against the theory as an absolute rule that cases of manic depressive and dementia præcox do not occur in the same family. At least one well-marked manic depressive woman was shown to have an undoubted dementia præcox brother and there were several other possibly similar cases, the diagnosis, however, not being so certain.

On the other hand, there are a number of instances of the same psychosis in the relatives both in dementia præcox and manic depressive.

ACUTE ALCOHOLIC HALLUCINOSIS (ACUTE ALCOHOLIC PARANOIA).*

By WILLIAM C. GARVIN, M. D.,
Assistant Physician, Manhattan State Hospital, New York City.

The characteristic clinical features which form the symptom complex of this group of alcoholic psychoses appear, as yet, to be imperfectly understood by physicians engaged in insane work. An examination of the yearly reports of the various insane hospitals throughout the country will bear out this statement. The literature in English on the subject is very meagre. This is rather surprising, as the psychosis is a fairly common one in all insane institutions. I have thought then that a review of the German literature, together with our observations at the Manhattan State Hospital, might prove of general interest.

To Wernicke belongs the chief credit for his extensive and exhaustive studies in delirium tremens. Korsakoff performed brilliant work in isolating that particular form of mental and physical disorder which bears his name. Ziehen, Wernicke and Krapelin have contributed to our knowledge of acute alcoholic hallucinosis; but it remained, however, for Bonhoeffer of Breslau to make the most detailed and accurate study of this type of alcoholic mental disorder.

The symptom complex under discussion presents the following characteristic features:

The sudden onset in an alcoholic individual of hallucinations of hearing of a persecutory character, with anxious fear reaction or silent brooding, without loss of orientation or memory.

The disorder, as Bonhoeffer states, exhibits many features which bear a strong resemblance to delirium tremens and, for a thorough understanding of their relation, an exact knowledge of the cardinal features of this psychosis is necessary.

This observer mentions a systematized form of delirium tremens with transition to acute hallucinosis. We receive a number of such cases from the alcoholic ward at Bellevue Hospital, but as no detailed records are kept in that ward, we are unable to give

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, June 1-4, 1909.

a complete history of the psychosis in its acute stage; and, therefore, we omit giving an example of such transitional forms. We, however, give below three fairly common types of the disorder under discussion.

TYPE I.—A pure auditory hallucinosis associated with intense fear. No systematization. Duration 10 days.

A woman, aged 31, bookbinder by occupation, was in the habit of drinking five to six glasses of beer and three or four glasses of whiskey a day.

One week before admission she became restless, irritable and sleepless; would start at slight noises and complained of ringing in her ears. She also heard voices say she was to be killed; thought that a murder had been committed in the house and spoke of killing herself.

On admission the patient was intensely depressed and apprehensive and reacted constantly to auditory hallucinations of a terrifying character. She heard men's voices say she would be killed; that her body would be cut up into pieces and burned, and heard the shrieks of her brother and mother outside the building as they were being murdered.

No hallucinations in other fields. No paræsthesias. Orientation and memory intact. The hallucinations subsided in three days. Insight complete.

TYPE II.—An acute auditory hallucinosis, associated with false sense perceptions in other fields. Incomplete systematization. Duration one month.

A clerk, aged 44, habitually drank four to eight whiskeys and several glasses of beer daily.

One month before admission commenced to hear vague sounds and indefinite voices which caused him worry and uneasiness. He ceased drinking and the voices left him. Three weeks later began drinking again and voices of a distinct threatening character appeared. They said he had committed a murder, stole a pocket-book, led a drunkard's life and threatened to kill him. He saw pictures and shapes on the wall, became frightened and applied to the police for protection.

On admission was intensely depressed and apprehensive. Spoke of hearing voices of both friends and enemies, coming from the walls and outside the building. The voices of his enemies would say, "Kill him. You will be poisoned. You have committed a murder and will be electrocuted. We will poison you." Friendly voices said, "We will save you. We will save your soul." He also believed the voices could read his thoughts, and spoke of a throbbing on the top of his head, which he thought was due to his enemies puncturing his skull with a needle. He complained of feeling fire on his elbows and heels and of ants crawling over his body. On account of these sensations he thought the X-ray had been used on him by the devil.

Visual hallucinations were present in the form of devils, shapes and pictures. He also thought that noxious gases, like sulphur, arose from the

ward. Orientation and memory were intact. He believed all these persecutions were inaugurated by enemies, but by whom he did not know.

After two weeks, the threatening voices disappeared. He, however, spoke of the voices repeating aloud what he had read. The paræsthesias left him after a week. At the end of a month all signs of the psychosis had disappeared. Insight was complete.

TYPE III.—Acute auditory hallucinosis with seeming disturbance of orientation and memory. Complete systematization. Fantastic delusions.

A laborer, aged 27, in the habit of drinking four whiskeys and seven or eight glasses of beer daily.

Two weeks before admission, he became irritable, suspicious and apprehensive; heard voices of his enemies say they were going to kill him; threw bottles out of the window in the direction of the voices; swore at them, and spoke of people watching and following him on the street.

On admission the patient was extremely suspicious, restless and apprehensive. Spoke of hearing men's voices say that they would kill him, mutilate him by cutting off his arms and legs and remove his brains and eyes. He mentioned an imaginary fight with the attendants, who wished to cut him up with a knife. At night he was very restless and apprehensive, and attempted to get out of the window in order to escape his enemies. He named two men with whom he had a quarrel one and a half years ago as being the instigators of his persecutions. They offered $50,000.00 to have him killed in order to be revenged.

He was uncertain as to the nature, character and location of the hospital and his time orientation, as well as his memory for recent events, appeared impaired. No insight.

For two weeks he continued suspicious, apprehensive and sleepless, muttered to himself, misinterpreted the movements of the attendants and several times assaulted them, thinking they were going to harm him. Would repeatedly refuse sedative, saying it was poison. Saw shadows on the wall and men being murdered in the ward; heard threatening voices coming from the bed and various portions of the walls.

At the end of the third week, the psychosis had disappeared. It was found that the seeming disturbance in his orientation and memory for recent events on admission was not real. Insight complete.

THE AUDITORY HALLUCINATIONS.

The essential and characteristic symptom presented by the psychosis is the auditory hallucinations. The voices may appear abruptly, with or without the preliminary phenomena mentioned below, or in a certain proportion of the cases the onset may be subacute and periods varying from a few weeks to several months may elapse before the voices become sufficiently annoying to bring about such a state of mind as to necessitate the commitment of the patient.

Frequently in the initial phase of the psychosis the patient exhibits an increased irritability to noise, slight sounds cause an apprehensive starting. Buzzing, roaring ringing or humming voices are often heard in the ears. These phenomena may continue on into the acute phase or they may disappear as soon as the voices become prominent, and then return again during convalescence.

During the active stage of the psychosis the auditory hallucinations are numerous. They are uniformly of a threatening character and engross the attention of the patient to the exclusion of all other interests. The following were the most frequent hallucinations observed in sixty cases:

The patient heard that he would be killed and cut to pieces; dismembered in various ways, burned alive, shot, poisoned, that his family have been tortured and murdered. Heard himself cursed, called a thief, murderer, bum, drunkard, no good; that wife or husband was unfaithful. Wherever he went, people said there he is, catch him—there he goes, or the voices of a gang continually threatened and followed him. Accusations of immorality and infidelity were noticed more frequently in the women than in men. Religious hallucinations, when present, are usually of a condemnatory nature. The voices said that the patient would be damned, sent to hell and his soul would be lost, etc. Grandiose hallucinations on which the patient builds expansive ideas are uncommon—we have seen only one such case.

The voices come from all directions. When asked where they come from, the patient usually points to definite locations, i. e., from the walls, floor, bed, telephone, ventilator, upstairs, below or outside the building. Now and then they speak of them coming by wireless or by auto-telephone. The voices are generally loud and distinct, and are uniformly those of his enemies, but occasionally the voices of friends giving him friendly warnings. The voices have a definite sound and character. They are those of men, women or children. Bonhoeffer states that sometimes voices in a peculiar unknown tongue are heard. A very characteristic feature is the hearing of a babel of voices, each voice apparently trying to drown out the other so that the individual voices cannot be made out. This author also mentions the frequent occurrence of voices which speak with a measured rhythm. Illberg and Wernicke report cases in which the patients hallucinated in

metrical form synchronous with the beat of the pulse. Mention is also made by authors of cases in which the patient hallucinated rhythmically to the tick of the watch or clock. These phenomena were not observed in our cases.

The voices are generally heard in both ears with varying degrees of distinctness. We have, however, seen several cases in which a change in the position of the patient influenced the intensity of the hallucinations; for example, when the patient lay on his right ear the voices were heard more distinctly on that side and seemed to come from the pillow. The patient usually hears single words or short sentences. Not infrequently he hears voices engaged in conversation about himself (Kräpelin). These are uniformly of a threatening or slanderous character. We have observed patients listening attentively to the conversation and answering the voices in angry profane manner.

An attitude of attention in many patients favors the appearance of voices. We have been able in a few cases by distracting the attention of the patient by rapid conversation and humorous remarks to cause a temporary cessation of the hallucinations. During the conversation the patient would laugh, smile and for the moment lose his fear affect.

Alternating voices are sometimes heard. One of our patients heard the voice of the devil say he would be killed and damned. The devil's voice would cease and he then heard the voice of an angel say he would be saved. Another patient spoke of hearing a man's and woman's voice alternately calling him all sorts of vile names.

Hallucinations of Sight.

These are present in a considerable number of cases, but play a subordinate rôle. The closer the relationship to delirium tremens, the greater the prominence of the visual hallucinations. Among the most frequent visual phenomena observed were pictures, shapes, statues, faces, heads of people, flashes of light and indefinite objects moving around the room.

Paræsthesias.

These are quite common. The patient feels an itching, burning, tingling or numbness in various portions of the body. They often

speak of these phenomena as electricity and say it is caused by their enemies.

Hallucinations of Taste and Smell.

These are not very common. Occasionally the patients will complain of odors like sulphur or some foul gas in the room, or of some odd taste in their mouths, which they often say is the result of some poisonous medicine they have been given.

Another frequent hallucination is that the patient believes his thoughts are repeated aloud but in the repetition they are distorted. We have had a patient who complained that the voices repeated what he had read or conversations with the physician. Another patient stated that the voices would ask him questions and if he did not reply, they would answer for him.

Imperative hallucinations are mentioned by Bonhoeffer. As a result of these, various motor manifestations are seen. We have seen two such cases.

Ideas regarding the viscera are rather infrequent. Ideas of reference and an anxious misinterpretation of ordinary occurrences are common. The patient sees a policeman or a crowd of people on a corner waiting for a street car and imagines they are looking at him. The nurses who casually glance in his direction are plotting to kill him. He refuses medicine and food because the nurse happened to make a chance remark or what he considered a peculiar gesture. He resists taking a bath for fear he will be drowned and starts suddenly in fright at any sudden movement in his direction, etc.

The Tendency to Systematize the Hallucinations.

Another characteristic of the psychosis is the tendency of the patient to bring his hallucinations into a connected delusional system. Physical explanations of the origin of the voices are common. They come from the telephone, hot air register, through the window from people outside the building or from people whose footsteps he hears and so on. Ideas of infidelity are developed from chance remark of the husband or wife. These are more common in women than in men. The person with whom the patient has had a recent quarrel is regarded as the one who is at the bottom of his persecution. More rarely he will refer back his

persecutions to such a remote period, prior to the onset of the hallucinations, that the physician will be in doubt whether or not he is dealing with a more or less fixed delusional state. We have had patients who named persons with whom they have had friction as remote as two to four years prior to the psychosis as being the instigator of their persecutions. The systematization, however, in the greater number of cases is superficial and changes constantly with the character of the hallucinations.

Ideas of wealth and grandeur are not common. In the one case in which they were present, they developed from the hallucinations, but were unimportant features of the psychosis.

The Characteristic Affect of Fear.

The behavior of the patient in his reaction to the hallucinations, is, next to auditory hallucinations, the most important feature of the psychosis. He is in continual fear. This is most pronounced during the acute stage of the psychosis. In the lighter cases, in which the hallucinations are not fully developed, there may be nothing more than an uneasy anxious dread. The patient, in the most severe cases, lives a life of torture. He becomes restless, irritable, uneasy and frequently moves about or even goes so far as to change his residence in order to get rid of the threatening voices. Quite a number go to the church for assistance or to the police for protection. In this irritable state, they are frequently assaulting or homicidal. Some attempt to end their troubles by suicide.

The fear affect is deep grounded and the patient cannot, as in delirium tremens, be persuaded out of his fears (Bonhoeffer). In many cases the fear reaction is manifested by an anxious hunted expression, or else they appear depressed, sit brooding over their troubles and are very irritable and uncommunicative. One of our cases presented a strongly stuporous aspect, seemed self-absorbed, spoke only after being urged and later committed suicide. Kräpelin mentions a mixed affect of half fear and euphoria. Bonhoeffer found this reaction in the transitory forms between delirium tremens and acute hallucinosis.

In many cases the fear subsides to a great degree upon their entrance into the hospital, although the hallucinations continue. They say they feel safer in the hospital for their enemies cannot reach them there.

Stream of Thought, Orientation and Memory.

The patients' stream of thought shows no alteration. Their orientation and memory remain intact. We have found that those cases in which the orientation and memory for recent events appear impaired, exhibit more or less delirious features. There is not, however, a real loss of grasp on his surroundings and time relations, as we found later in the course of the psychosis that the patients possessed a surprisingly accurate knowledge of all that transpired during the period in which an apparent disturbance seemed evident. The closer the relationship to delirium tremens the more disturbed is the memory and orientation. Sleep is always disturbed during the acute stage as the hallucinations are usually more vivid at night.

Physical Signs.

The physical signs which accompany the psychosis are unimportant. Tremors of the hands and tongue, gastric disorders and frequently slight neuritic symptoms are present. Bonhoeffer states that an acute hallucinosis with polyneuritis and amnesia is occasionally seen. Vaso-motor disturbances, as sweating and cardiac palpitation are sometimes present. Albumin, as a rule, is not found in the urine.

Progress Toward Recovery.

The return of the mental symptoms to normal conditions takes a longer time than for the development. First of all there is a disappearance of the fear affect. Parallel with this the hallucinations becomes less prominent. Residuals in the form of voices, ringing, buzzing or humming in the ears or a roaring in the head often persist for weeks.

During convalescence, reappearance of the voices is very common, and when this occurs there is a return of the affect of fear, either open or, as is often the case, in the form of silent brooding.

The sudden appearance of insight is one of the remarkable features of the psychosis. The patients have an excellent memory for occurrences during the acute phase of the disorder. An extended investigation of the patients' mental states during this period will frequently yield remarkable results. The wealth of

their hallucinations and delusions as well as the fantastic character of their experiences are astonishing. They are much more numerous and varied than one would suppose from mere observation of the patient.

Duration.

The duration of the psychosis varies between a few days and several weeks. A considerable number of our cases, however, have been prolonged for months. Cases are reported which have run for over a year with finally complete recovery. Relapses are common and frequently occur after days and weeks during which the patient is free from hallucinations. Residuals in the form of humming, buzzing or roaring noises in the ears or head are common and cause considerable annoyance to the patient.

The sullen, depressed, irritable and uncommunicative cases appear to run a longer course than the frank accessible ones. Their conduct is frequently marked by sudden outbursts of temper and violence with profane and abusive language. Sudden assaults on fellow patients on account of chance remarks or in response to their hallucinations are very common. These patients often deny hallucinations, yet judging from their conduct, it is evident that they are still under their control.

Apart from this group, a number of cases, after the disappearance of the hallucinations, exhibit an irritable and unstable mood. They are very persistent in their demands for release and show marked resentment against the physician on account of their detention.

Bonhoeffer states that the duration of the psychosis depends upon the closeness of its relation to the delirium tremens, that is, the greater the tendency to disorientation and combined hallucinations, the shorter is the course of the psychosis.

Prognosis.

All writers give a favorable prognosis for the first attack. A patient, however, who has suffered from an attack of acute hallucinosis is especially prone to have further attacks of alcoholic insanity if his excesses are renewed. The second attack is almost certain to be in the nature of an acute hallucinosis if over indulgence follows soon after his discharge from the hospital. These

patients are especially vulnerable to the effects of alcohol; very small quantities are sufficient to cause another upset. Other contributory factors in these cases are worry over inability to obtain employment, family quarrels, lack of proper food, accidents, the infectious diseases, etc.

In our list of sixty cases we have had nine who developed a second attack, and one who is now in the hospital under treatment for his fourth attack.

The prognosis, as regards the life of the patient, depends upon the time the patient is placed under close surveillance or brought to the hospital. The tendency to suicide is very great, but the danger, however, does not end with their commitment. These patients form one of the largest groups of acute psychoses with suicidal tendencies, and give hospital physicians much concern as regards their safety. Repeated attempts at suicide are not infrequent, and in our cases occurred more frequently in women than in men.

A striking illustration of the tendency of these patients to end their troubles by suicide is shown by the fact that among the sixty cases we examined (twenty-four women and thirty-six men) six men and seven women attempted to take their lives by various means.

Death from intercurrent diseases is not nearly so common as in delirium tremens.

Etiology.

Prolonged alcoholic excesses produce the psychosis usually in conjunction with recent over-indulgences. All the patients personally examined by the writer were both beer and whiskey drinkers. In a few especially susceptible individuals, however, smaller quantities may cause a mental upset. The disease attacks a better class of individuals than those who suffer with delirium tremens. The largest number of our cases in men were among trade workers and in women among those who were married. The preferred age is the third decade of life. Cases are uncommon in the early twenties and over forty-five. Of our sixty cases, 32 were of Irish birth, 14 born in the United States, and the balance divided among seven European States. The Jewish race is conspicuous by its absence. Other contributory factors are often

present, especially in unstable individuals, fright, shock, quarrels, over-work, accidents, diseases, like pneumonia, influenza, or tuberculosis.

Diagnosis.

A searching anamnesis from the patient and his friends or relatives is of the greatest importance. Definite information should be obtained as to the exact quantity, frequency and form of alcohol imbibed. This is of the utmost importance in enabling us to differentiate hallucinoses of non-alcoholic genesis from alcoholic disorders, and also aids us in forming an opinion in those cases in which alcoholic features temporarily mask or play a part in bringing into prominence other mental conditions, for example, paranoid dementias.

The patient and relatives should be carefully questioned with reference to previous acute hallucinatory episodes with fear reactions. The former attack may have only been in the nature of a slight uneasy apprehensiveness without hallucinations, or the latter may have been incompletely developed. Patients often continue at their vocations during the lighter attacks and forget to mention them unless closely questioned.

Exact information regarding the patient's make-up is another important feature of the anamnesis. Dr. August Hoch, in his study of manic-depressive insanity and dementia præcox, has shown us the value of such knowledge. It is of especial value in cases which present features which awaken a suspicion that we are dealing with something more than an acute alcoholic hallucinosis.

The diagnosis of acute alcoholic hallucinosis in most cases offers no difficulty. The history of prolonged alcoholic excesses; the sudden appearance of hallucinations of hearing of a threatening, annoying character associated with fear, apprehension or silent brooding; the rapid development of ideas of persecution and harm, together with the preservation of orientation and memory, form a symptom complex that is not easily mistaken.

In delirium tremens, hallucinations are chiefly in the optic and tactile fields; there is disorientation, disturbance of memory for detail and for time succession of occurrences, florid confabulations and disturbance of perception. Moreover, the course is short, usually one week.

Acute hallucinoses of non-toxic origin are occasionally seen which present the same clinical features as acute hallucinoses of alcoholic genesis. We have under observation a number of such cases in which fright, shock, worry, privation and fears of being robbed, etc., appeared to have been the etiological factors. Alcohol could be definitely excluded.

The diagnosis of acute alcoholic hallucinosis from the paranoid form of dementia præcox occasionally offers difficulties, especially if the patient has recently over-indulged in alcohol; here, the alcohol may have been a large factor in bringing into prominence the fundamental disorder, or alcoholic features may dominate the picture to such an extent that the diagnosis for the time being may be in doubt. Bonhoeffer states that when intestinal hypochondriacal complaints or taste paræsthesias are present and form a prominent feature of the psychosis, we may be sure that we are dealing with something more than an acute hallucinosis. Kräpelin regards the hearing by the patient of voices engaged in conversation about himself, to which he is an attentive listener, as of diagnostic value in acute hallucinosis. In doubtful cases all the facts should be present before proceeding to a definite diagnosis. We will then have little difficulty if the cardinal features of the psychosis under discussion are borne in mind.

Toxic psychoses resulting from the over-use of morphine, chloral, cocaine, trional, veronal and other hypnotics, are frequently accompanied by hallucinatory states with fear and apprehension. Many of these patients are heavy drinkers and thus alcoholic features may complicate the situation. These cases should be grouped according to which toxic features dominate the picture. In the drug psychoses we have a delirium with clouded sensorium. Hallucinations of sight, as well as those of hearing, are prominent; the latter being usually of a dream-like character. Tactile hallucinations are common, especially in cocaine delirium. There is great fluctuation in the mood, the fear being scarcely ever continuous. In addition there is disorientation, confusion, rambling or frequently incoherent utterances. On recovery there is usually amnesia for the acute phase of the disorder.

With reference to progressive mental disorders developing after acute alcoholic hallucinosis, there seems to be a difference of opinion. We have had a number of cases in which the symptom

complex at the initial examination appeared sufficiently characteristic to warrant the diagnosis of acute alcoholic hallucinosis, but the subsequent course of the psychosis made it plain that we were dealing with a more chronic form of insanity. We are collecting a number of such cases for future consideration.

REFERENCES.

Wernicke: Grundriss der Psychiatrie.
Ziehen: Psychiatrie.
Kräpelin: Klinische Psychiatrie.
Bonhoeffer: Akuten Geisteskrankheiten der Gewohnheitstrinker.
Osler: System of Medicine.

A REPORT OF THREE CASES OF KORSSAKOW'S PSYCHOSIS.*

By CHARLES E. STANLEY, M. D.,
Assistant Physician, Connecticut Hospital for the Insane, Middletown, Conn.

In the last twenty years, since Korssakow first published his very clear and comprehensive description of the symptom-complex of polyneuritic insanity, the number of reported cases of the psychosis which bears his name has grown into the hundreds. Some confusion concerning the disease arose, at first, even in the minds of careful observers, and many cases were published which were lacking either in the characteristic physical or mental symptoms of the disease.

Naturally, with the passage of time and the accumulation of reported cases, the observations of Korssakow have been enlarged upon and the clinical aspects of the psychosis have become greatly varied. Running through all the various descriptions of cases, however, there appear certain cardinal symptoms which have come to be recognized as essential to the disease, the mental picture of which has now become quite clear.

The disease is usually sudden in its onset, with insomnia, restlessness, delirium and hallucinations. Sometimes it begins with stupor and hallucinations and less frequently develops gradually from a condition of chronic alcoholism.

Korssakow, in describing the psychosis, made two large divisions. In the first he included all cases ushered in or accompanied by delirium. In the second, he placed all the forms characterized by confusion or stupor. Dupré, who, in Paris, examines all prisoners in the city suspected of insanity, described the disorder under five heads, while Knapp in his excellent monograph goes still further and makes no less than eleven different divisions. The latter recognizes a delirious form which is present in at least two-thirds of all persons suffering from this psychosis. Both Dupré and Knapp describe a stuporous or confusional form. For

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

all practical purposes, however, the divisions proposed by Korssakow are sufficient.

The disease is due to a toxæmia. The toxic agent in the great majority of cases, 75 per cent, according to Bonhoeffer of Heidelberg, is that seductive poison which has been the boon companion of man in all ages—alcohol. The psychosis occurs, according to the same authority quoted above, in 3 per cent of all chronic alcoholics. It is much more frequent in women than men. In eight cases observed by the writer, all, with one exception, were women. The quiet, steady drinkers are the greatest sufferers, in whom the existence of the alcoholic habit is frequently unsuspected until the disease appears. The chronic poisoning of the system by alcohol, then, either per se or by causing the production of some other toxic agent in the body, is the most prolific cause. Some authors consider it the sole cause, and look upon the disease as simply an aftermath of delirium tremens. The metallic poisons, carbonic acid, syphilis, tuberculosis, sepsis, severe anemia, carcinoma, trauma, fever, etc., are all enumerated as causes of the symptom-complex. It is possible that tuberculosis may at times be secondary to the disease in question.

Roemheld (of Honegg) in *Archives of Psychology*, Bd. 41, No. 2, reports a case of *brain syphilis*, with Korssakow's symptom-complex, cured by anti-syphilitic treatment.

Doctors O'Malley, Franz and Blackburn, of the Government Hospital at Washington, publish a case of polyneuritis of *auto-toxic* origin with Korssakow's syndrome, and with report of autopsy and microscopical findings.[1]

Multiple neuritis furnishes the prominent physical symptoms, hence the name polyneuritic psychosis, and either precedes or is concomitant with the delirium which frequently ushers in the disease. It more seldom *follows* the delirious condition. All the nerves of the body are probably affected. Pain in the extremities with tremor and loss of power, particularly in the extensors of the hands and feet, causing the wrist and ankle drop, follow in short order. Occasionally there is also paralysis of the occulo-motor, facial and pneumogastric nerves, muscular atrophy, diminished electrical reaction, clonus, loss of deep reflexes, paræsthesia and

[1] American Journal of Insanity, October, 1908, Vol. LXV, No. 2.

hyperæsthesia, in the paralyzed parts; nausea, anorexia, vomiting; rapid, feeble, irregular and weak pulsations of the heart. Occasionally, also, there may be at the outset epileptiform or apoplectiform seizures; speech disturbances and Romberg sign.

Disturbances of the mind are evidenced by:

1. Hallucinosis—aural and visual illusions or hallucinations.
2. Amnesic disorientation.
3. Impaired memory for recent events, with confabulations, falsifications and pseudo-reminiscences.
4. Hyper-suggestibility.

The hallucinosis is of no definite duration. The most marked mental symptom of the psychosis is the peculiar memory defect—the markedly diminished impressibility of memory, as a result of which disorientation is usually complete. Time, place and events are hopelessly jumbled. Gaps in memory are filled in with fabrications and confabulations. Memories of the hallucinatory delirious state are retained after the hallucinations have subsided.

It is in the field of memory that the most marked changes are noticed in this disease. Events of one minute are forgotten in the next. Memory is affected under all three of the divisions given by Ribot, *i. e.*, retention is impaired as well as the power to recall and localize.

"There is a disturbance of function, a failure to assimilate impressions from without into consciousness and to represent those before it which have already been assimilated in the past—in other words, a failure of mental synthesis."[3]

Fabrications occur in which actual events are hopelessly mixed and described in absolutely "false sequence and connection"—leading to fantastic stories. There may be also "hallucinations of memory" in which patients confidently recall events which never happened. To this latter, Wehrung would limit the term "pseudo-reminiscences," while he uses "confabulations" for the fabrications a patient uses to bridge over awkward gaps in his memory. Some writers go so far as to say that pseudo-reminiscences are essential to the existence of the disorder, and the more pronounced these hallucinations, the deeper the disturbance of ideation. The emotional condition is subject to oscillations and

[3] F. W. Mott, Archives of Neurology, Vol. III.

greatly influenced by suggestion. The patient is generally cheerful and sometimes jocular, but underneath this is a condition of peevishness and irritability.

In the stage of delirium, the more profound clouding of consciousness, and the character of the hallucinations and illusions serve to distinguish it from the post-infection psychoses The prognosis in the latter is also much more favorable.

In paralytic dementia, the onset is gradual with greater speech disturbances, marked paragraphia and cerebral paralysis. The memory of the alcoholic is more affected for recent events, of the paretic, for more remote happenings. In presbyophrenia, there is absence of alcoholic history and neuritic disturbances, a childish emotional state and a sort of busy restlessness, especially at night. The age of the patient, also, with arterio-sclerosis may help to differentiate, but the difficulty may prove greater if the presbyophrenic has had an alcoholic history.

Prognosis is unfavorable. The disease rarely, if ever, terminates in complete recovery, and may prove fatal in its early or later stages. The course is apt to be prolonged, resulting, in the most favorable cases, in more or less impairment of memory and emotional deterioration.

Pathology. The hemorrhagic polyencephalitis, described by Wernicke, in cases dying in the early stages, is what one would expect to find in severe alcoholic intoxication (Kraepelin). The whole nervous tissue from cortex to periphery is involved in destructive changes. In the cortex degenerative changes are noted in the tangential fibers and in the spinal cord, atrophy of the fibers, particularly of the columns of Goll.

The treatment consists in the withdrawal of alcohol and suitable surroundings either at home or in an institution, of rest in bed and on an air bed if bedsores threaten. In the early stages the treatment is identical with that of delirium tremens. The neuritic symptoms are alleviated by hot applications and anodynes, gentle friction, at first, followed by massage and electricity later, to prevent atrophy of the muscles. Some authors advocate the use of fats and the administration of strychnine.

In the ten-year period between 1898 and 1908, there were four thousand, three hundred and seventy-seven admissions to the Connecticut Hospital for the Insane. Five hundred and forty-two

or about 12½ per cent of this number were of toxic origin, and five hundred and thirteen of the latter number, including eight cases of Korssakow's psychosis, were grouped among the different forms of alcoholic insanities. Each of the eight cases of Korssakow's psychosis, mentioned above, had well authenticated alcoholic histories. Three of these were admitted to the hospital between April 30 and September 23, 1908. A description of these cases, two of which are still under observation, follows:

Case I.—W. H. D. Born at Batavia, N. Y., in 1867, of English and Irish parentage. Married; developed normally, received a common school education, learned the carpenter's trade, and later worked as brakeman and trackman on railroad. Last worked for N. Y., N. H. & H. R. R., at Bridgeport. Habits have been very intemperate for years; he admits the steady and excessive use of alcoholic stimulants and claims that he worked until his employers noticed his mistakes due to forgetfulness, and laid him off. Committed to the Connecticut Hospital for the Insane, April 30, 1908. Duration of disease unknown.

Physically.—Paraplegia, with ankle drop, knee and ankle jerk abolished. Fingers are partially flexed, stiff, shiny and markedly painful on forced extension. Pain in feet, legs and forearms, and over nerve trunks on deep pressure at bend of knee and elbow. Loss of power shown by inability to stand or write. Galvanic irritability lessened. Pupils even but sluggish, tongue protruded in median line, tremulous; tremor of eyelid and facial muscles. Heart pulsations accelerated by slightest exertion, sounds rapid, irregular and weak, probably due to involvement of the vagus. Pulse 90, soft. Romberg sign present. Test words pronounced correctly. Urinalysis shows marked indicanuria.

Mental Symptoms.—Attention is easily held. He comprehends questions, is disoriented for time and persons, and varyingly so for place. He is confused and manifests marked disturbance of impressibility of memory. He recognizes his physician and nurses, as such, but, although he has been frequently told, he is totally unable to recall their names. Memory for events of psychosis is strikingly impaired. Asked, "How long have you been in this place?" replied, "four months" (really three weeks). Almost immediately he was again asked, "How long have you been here?" "Since early last fall," apparently having forgotten his last answer. He then begins to talk of himself, "I worked for the N. Y., N. H. & H. R. R., at Batavia—no Rochester," etc., not realizing that Batavia and Rochester are not on the N. Y., N. H. & H. R. R. "Came here with a hackman, only, from Batavia—no—Rochester—no—Bridgeport. I ran away from the hospital and I led them a chase," etc. This last is a pseudo-reminiscence, probably of his delirious state while in Bridgeport Hospital. He manifests no true insight into his condition, attributing his illness wholly to rheumatism. Emotionally, he is exceedingly unstable, usually cheerful, smiling and

responsive, but influenced by suggestion, quickly becomes irritable, sad and tearful.

July 24, 1908. No hallucinations of any type have been noted since admission. Orientation is still impaired for time, but correct for place and persons. He knows that he is in the Connecticut Hospital for the Insane, but is unable to tell the date of his entrance or how long he has been here. As a result of his memory defect he is still confused and contradictory, and mixes dates, events and places. He does not fabricate.

October 1, 1908. Now dressed and on the ward. Appears clearer, realizes his mistakes and tries to correct them. He states that, to the best of his knowledge, he worked on the Cos Cob division of the N. Y., N. H. & H. R. R. last winter. "I was in the habit of drinking, which often incapacitated me for work. At last I laid around saloons drinking and stopped working altogether. I must have been sick or insane for I was sent to the Bridgeport City Hospital (almshouse) by my lodge of Red Men and I think in a month or so I was sent here." He is not clear as to date of entrance to or length of stay in the Bridgeport Hospital. He realizes his mental weakness and his inability to work and bemoans his fate. He tries hard to fix dates and reconcile matters.

October 28, 1908. His gait is still awkward and unsteady, but improved. Mentally, practically the same as last noted. Memory defect still apparent. Discharged to-day to custody of friends as improved. In hospital six months.

Case II.—Mrs. I. H. A poorly-nourished woman, thirty-nine years of age, native of the north of Ireland; Protestant; has been twice married and is now divorced; has had three children, all of whom are dead except one by her first marriage. Her early occupation was that of dressmaker. Of late years she has rented a house, and let rooms for questionable purposes. No history of neuroses or psychoses traced in family. Syphilis and trauma denied. Negative menstrual history.

Habits.—For at least a year, and probably for a much longer period, she has been addicted to the excessive use of beer, gin, whiskey, tea and coffee.

Admitted to the Bridgeport City Hospital July 30, 1908, complaining of pains all over the body, deep-seated and boring in character. Onset was sudden. She had no chill, sweats or elevation of temperature, but vomited considerable bile, was constipated, had severe pain in epigastrium, anorexia, and eructations of gas. She also suffered from palpitation, dyspnœa and precordial pains. Heart sounds were weak and floppy, aortic accentuated, deep reflexes were absent, pupils sluggish. There were no areas of anæsthesia or paræsthesia. Muscles of hands, forearms and legs were weak and somewhat atrophied with beginning contractions. Dropping of wrists and feet was well marked, trunk muscles also involved; some edema of feet; pain or pressure over nerve trunks. She was unable to use her hands or to walk and general debility was so marked that she was placed in bed.

Mental Condition.—Illusions of sight and hearing were numerous, but very changeable. Her language was obscene and profane, with marked and changing fabrications and confabulations. She was disoriented for time, place and persons.

Brought to the Connecticut Hospital for the Insane two months later, September 23, 1908, and taken to ward on a stretcher; condition untidy. She had no use of her feet and legs; was unable to straighten her legs from knee down. The right leg above the ankle was somewhat discolored. She was unable to hold anything in her hands. Wrist and ankle drop was pronounced. Clonus of foot obtained by simply lifting the leg. Some edema of hands and feet. Pronounced tremor of fingers, facial muscles and eyelids. Legs were smooth, shiny, flexed and contracted, knee reflexes absent, pupils notched and reacted sluggishly. Pain elicited on pressure over nerve trunks.

October 2, 1908. Habits untidy and filthy. She is restless, uneasy and talkative, especially at night. Fabrications and pseudo-reminiscences are still prominent.

October 13, 1908. Lumbar puncture made to-day with the following results:

PRESSURE.

At the beginning of puncture	70 mm.
At the end of puncture	40 mm.
Variations	30 mm.

FLUID EVACUATED.

Amount	17 cc.
Appearance	slightly bloody; centrifugated until clear
Sp. gravity	1.006
Reaction	slightly alkaline
Reducing body (sugar)	trace
Protein	0.005
Potassium mg. in 10 cc.	2.500

November 2, 1908. Some disturbances of perception; she has illusions of sight and hearing; hears the voice of her daughter who has been dead for over a year, and sees her in the doorway clad in a checked dress. She has no conception of place and no stable idea of time. Mistakes persons about her for friends and relatives long since deceased. She has no insight, but expresses no delusions. Memory for striking events of her past life, such as her marriage, births and names of her children, death of her daughter, is retained, but the dates of their occurrence are unknown. There is also marked amnesia for all of the events of the psychosis. Pseudo-reminiscences, which may be enlarged upon by suggestion, in which she narrates improbable stories in a jocular way, are frequent. She laughs heartily over her fancied experiences.

November 12, 1908. No marked change. Shows the same impairment of memory. Orientation is imperfect and she has no conception as to the length of time she has been in the hospital. She recalls the death of her daughter, but cannot approximate the date of her decease. She is less chatty, and falsifications are less pronounced. She volunteered the remark that her daughter had appeared to her in the doorway and conversed with her.

December 15, 1908. She is now oriented for place and nearly so for time, but mistakes identities of those about her. Says she has been here three or four weeks (really three months). "I came here the last of November" (September 23, 1908). "Dr. T———, who roomed up in the village, came to me on Main St., Bridgeport, and asked me if I would not like to go away. He came with me, took my clothes from me and left me here to be treated for rheumatism." There is still marked amnesia for events of psychosis.

Content and Train of Thought.—She is loquacious. "I wish I had the 'tism' out of my bones. Have had it now for two years—no—going on two years—well it is more than one year. My memory is at fault because I am so nervous and easily rattled. After my daughter died a year ago, I kept a little store and worked in a laundry." Denies her drinking habits. "Oh, I have tasted it when it was going round, I took some—drank with others." Falsifications and confabulations of memory are very pronounced. She thinks she has been here a month (really six weeks). She makes a statement and immediately makes another entirely at variance with it, apparently having entirely forgotten her first statement. This peculiar memory defect was the most marked feature of the psychosis. She not only has pseudo-reminiscences, but also reminiscences of these falsifications, and also for more remote events. She answers some questions accurately, showing fewer lapses and fabrications than at first, and appears to be trying to correct some previous impressions. Thinks now that the voices she heard talking to her about her daughter may have been imaginary voices. Emotionally, cheerful, jolly, humorous—is "happy that she is living." Still has wrist drop, but can extend index finger of right hand fully and the others partially; pain in hands, forearms and legs and some hyperæsthesia—marked clonus of foot—knee-jerks abolished; tremor and ataxia.

June 1, 1909. Same condition prevails. There is considerable atrophy of extensor muscles of left hand.

Case III.—M. L. Born in Bridgeport, Conn., of Irish parents, thirty-three years of age. Married; shop employee in cartridge works. Developed normally; received a common school education, but never cared for study. Father was very intemperate. No history of insanity or neurotic tendencies in the family. Patient was married in 1898 and a son was born January 13, 1899, which lived only a short time. Four years ago her husband left her and since that time she has consorted with other men and led a dissolute life. For seven years she has been intemperate, and her

mother writes she has had syphilis. Has never had delirium tremens. The present is the first attack of mental trouble, and was of sudden onset, about four months ago. Speech became thick; gait, unsteady. She became talkative and restless, and at other times dull and apathetic; suffered from insomnia. Habits were filthy. Memory was poor for all events. She rambled in her talk, was mildly delirious, saw people who were not present, handled an imaginary child, feeding it candy, and complained of getting the candy in her hair. Passed urine and feces in bed. She was entirely unconscious of her surroundings. Although her whole body was more or less paralyzed from alcoholic polyneuritis, and she had passed her urine and feces in bed, she declared that there was nothing the matter with her. She sang and talked at night and thought she could get up and walk. Imagined the attendant was her husband.

Admitted to the Connecticut Hospital for the Insane, June 24, 1908. Aural and visual hallucinations cannot be elicited. She mistakes attendants for former acquaintances. Is completely disoriented. Says the season is fall (June, 1908). In reply to questions as to place, she says she is in a boat, then remarked, "No, this is my home at Stratford, Conn." She comprehends questions, but recent past events cannot be recalled; says it is morning (really 4 p. m.). She shows retrospective falsifications of memory. She cannot tell her age, when she went to school, nor the date of her marriage. Fabricates—says she slept in the corner bed—but arose this morning and got into the one she now occupies. Says she sees her husband every night. "Last Thursday or Friday—I just forget now what day it was—we took a trolley ride up to New Haven and had a fine time; then went to Savin Rock and stayed there a few hours and had dinner, and then came home." Asked about the dinner, she replied, "Oh, we had a fine dinner—about ten courses—and everything you could imagine to eat. But we did not go right home, but went to Seaside Park and sat out on the benches for about two hours." Says her father has died since she came here, she received notice of it yesterday and went to make arrangements with her mother this morning in regard to the burial. "I have seen you before with your wife, in New York, week before last when I was down there with Maggie. You treated me for rheumatism." In the emotional field she is subject to marked oscillations. As a rule she is happy and cheerful, but easily irritated and angered.

Physical Symptoms.—Quadruple paraplegia, from motor and sensory polyneuritis; wrist drop; ankle drop; pain and hyperæsthesia in affected areas, and over nerve trunks on pressure; some atrophy of extensors; pupils dilated and very sluggish to light and accommodation; fine tremor of tongue; chronic nephritis; impaired speech, gait, station, and general muscular weakness.

August 10, 1908. Still completely disoriented. Says she is in a boat, "Came here this morning in a boat from New York—this is a boat and we will be in Bridgeport in a little while. I went to New York last night and left my pocket-book there. I went to a show with another girl." Content of

thought is made up of fabrications and pseudo-reminiscences. She laughs and converses with Case II, whose bed adjoins hers, and seems very jolly and happy.

August 21, 1908. Polyneuritic symptoms not so marked, especially on right side, in both arm and leg, and wrist drop not so prominent. Speech not perceptibly changed. Cannot pronounce clearly the usual test words. Amnesia and pseudo-reminiscences continue. She now expresses fantastic and somatic delusions similar to those of outset. She felt of the counterpane and when asked what was the trouble, said, "Why, this is a dog skin and must have come from that dog I felt on me."

Lumbar Punctures Made:	Sept. 9, 1908.	Oct. 13, 1908.
	PRESSURE.	
At the beginning of puncture	150 mm	130 mm.
At the end of puncture	No more fluid could be obtained	60 mm.
Variations		70 mm.
	FLUID EVACUATED.	
Amount	4½ cc	32 cc.
Appearance	perfectly clear	perfectly clear.
Sp. gravity		1.006
Reaction	slightly alk	slightly alk.
Reducing body (sugar)		trace
Protein	1%	0.013
Potassium mg. in 10 cc		2.100
		46 CELLS IN 100 FIELDS.
Cells		32 lymph. — 69.57% 1 fibro. 2.17% 13 Endo. 28.26%

June 1, 1909.—Patient is now dressed and sitting up, and is much improved physically. She is still unable to pronounce test words, shows considerable impairment of memory, and is at times irritable and peevish.

SCARLET FEVER AS AN ETIOLOGICAL FACTOR IN THE PSYCHOSES.*

By EDGAR B. FUNKHOUSER, M. D.,
Assistant Physician, State Hospital, Trenton, N. J.

A glance at the evolution of psychiatry is sufficient to impress one with the fact that etiology has held a prominent place with psychologists from the mythical era to the present time. It is also noticeable that at different periods, different causes were considered sufficient to produce a psychosis. Certain forms of insanity have come down through the ages unchanged, but etiology has oscillated from the heavens to its antipodes. With this oscillation of the real or supposed cause, care and treatment has followed in a most natural order of sequence. When the aberrations were attributed to the smile of the gods, the favored one was looked upon as supernatural and venerated. When possessed of the devil, praying, singing of hymns, violent exorcisms and magical formulæ prevailed. Only when the etiology came within the confines of physical causes and human reason can we discern any real progress. It is said that Galen, the last of the great ones of the golden age, who wrote at the beginning of the third century, did credit to a twentieth century text-book. During the dark ages which followed, the science of medicine degenerated into an elaborate system of empiricism and mysticism, and demonology was reinstated as a controlling element in life. "The world seemed to be like a large mad-house for witches and devils to play their antics in."

We foster the modern view: a human being sick; physical causes; a hospital, light, airy, clean and comfortable; suitable and abundant nourishment, trained nurses, kindness, non-restraint, reason, liberty. But let us not be deluded. "The cost of giving the treatment has been materially increased, the recovery rate remains unchanged" (Bancroft).

The scientific study and treatment of the insane has met with signal achievement, but the results are far from gratifying.

*Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

The ever increasing percentage of insanity tends to turn our attention to that genius, Dr. Pliny Earle, of 30 years ago, who said, "Very clearly, if insanity is to be diminished it must be by prevention and not by cure." The old adage is also quite appropriate: "He who cures a disease may be the skilfullest, but he who prevents it is the safest physician." As *etiology necessarily precedes prophylaxis,* so a careful study of each *known* and *probable* cause seems worthy of consideration.

Where scarlet fever originated is not known; descriptions are given of it by medical writers before the Christian era. To Sydenham we owe the name and its differentiation from measles. It is very generally disseminated, it has been studied in all parts of the civilized world. No race is immune. Whether it is due to the streptococcus scarlatinæ of Klein and Gordon, or the streptococcus conglomeratus of Kurth, or an organism resembling the meningococcus as isolated by Wadsworth, has not been determined. Mallory would seem to demonstrate that the cause of the disease is probably a protozoön. Age does not confer immunity; susceptibility, however, is in inverse ratio to the age, the greatest incidence being from four to eight years of age.

Hospital care of scarlet fever cases has greatly reduced both morbidity and mortality, 23.48 and 1.56 respectively, per ten thousand population, in the Boston City Hospital. The mortality for the State of New Jersey during the past five years is given as 2.49 per ten thousand. The percentage of mortality varies, with age and epidemics, from 33 per cent to 1.33 per cent. The death rate in the Municipal Hospital of Philadelphia in 5213 cases was 9.72 per cent.

Although the mortality and morbidity has been considerably reduced, yet with hospital care and modern treatment, complications and sequelæ that may lead to permanent physical or mental impairment continues very high, estimated at over 90 per cent in some epidemics. Albuminuria accompanies nearly all cases of severe scarlet fever, often with interstitial changes especially marked. McCullom finds a mitral systolic murmur in 187 of 1000 cases analyzed. Otitis varies from 10 to 75 per cent. This in turn has been the direct cause of 10 per cent of deaf-mutism in the State of New York, in Great Britain 23.5 per cent.

The nervous system also receives no little portion of the damage

done by this disease. Convulsions are not infrequent at the onset of the disease in children, which indicates a sudden and severe disturbance of the physical equilibrium, or they may occur late as a result of uremia. It may be well to state in this connection that in many cases of epilepsy the initial convulsion occurred at the time of an attack of scarlet fever. Weildermuth reports 12 cases in a series of 187.

"More cases of epilepsy are consecutive to scarlet fever (apart from the influence of nephritis) than all the other acute diseases put together." "This together with the fact that optic neuritis may follow scarlet fever, without any organic change in the brain to cause it, seems to show that the scarlet fever poison has a special action on the nervous system" (Gowers).

The writer has found, in a series of 122 cases of epilepsy, 11 (more than 9 per cent) with a history of scarlet fever, the initial convulsion occurring at the time of the disease or as a result of some complication or sequelæ. Other factors, however, such as heredity, alcoholism and imbecility, were not excluded.

Meningitis, paralysis, embolism, hemiplegia and chorea have also been reported during the past year.

In regard to the relation of scarlet fever to the psychoses: The author has reviewed the literature at his disposal, but has failed to find much that treats it specifically, and no statistics bearing upon the subject except in an indirect way. It is also difficult to handle the information that has been gathered in a manner that would benefit those not already familiar with the subject. At the present time it shall be the attempt of this paper to bring together only such isolated facts as seem worthy of consideration; and if, by so doing, I assist anyone in any manner or shall have offered a suggestion that will lead toward the establishing of scarlet fever in its proper place as an etiological factor in the psychoses, then compensation will have been sufficient.

The question might well be raised here. Does the specific poison (or organism) of scarlet fever have a predilection for the nervous system, or, are its toxins more apt to affect the nervous system than the toxins of any other acute infectious disease?

To the first part of the above question with the light of modern research no definite answer can be given. Some would lead us to the view that scarlet fever poison has a special action upon the

nervous system, while others prefer to explain its attack upon an anatomical basis—continuity and contiguity of tissue. The former view seems to receive some support by the fact that optic neuritis may follow scarlet fever without any organic changes in the brain to cause it.

To the latter part of the above question there is also some ground for discussion. For those who would oppose the view that the specific poison (or organism) of scarlet fever has no predilection for the nervous system, there must be some explanation of its effects other than anatomical relations, especially in those cases in which no complications were recognized, the patient, apparently, not having had a severe attack. It is not uncommon to get a history of an ordinary attack of scarlet fever followed by a change in disposition, defective memory or arrested development. Furthermore it has been observed that more cases of nervous disease are consecutive to scarlet fever than all the other acute infectious diseases put together.

In dealing with the psychoses alone, it seems most natural to place them in two groups. First, those in which scarlet fever is a *direct* cause; and second, those in which it is an *indirect* cause.

In the first group are placed such forms as manic-depressive insanity, fever delirium, post-febrile psychoses, exhaustive psychoses and dementia præcox.

The first form is passed briefly as it occurs but rarely. The second form does not differ essentially from the fever delirium caused by the toxins of other acute infectious diseases. It follows rather closely the clinical course of the fever, and in a measure depends upon it. The clinical picture presents four different grades corresponding to the intensity of the toxic action upon the cortical neurons, varying from moderate irritation to paralysis and finally to complete destruction of the same. This form of psychosis is of short duration and in favorable cases subsides with the temperature. A few cases emerge from the fever delirium into an exhaustion psychosis or become the starting-point of other psychoses.

The third form of mental derangement in which scarlet fever is recognized as a direct cause is that known as post-febrile psychosis. This, as its name indicates, is found usually following the fever and is apt to lead to permanent mental impairment. Because of

this tendency some of its symptoms deserve mention. The mildest form is represented by those cases in which, after the subsidence of the fever, the patients fail to show their former physical and mental energy, they are dull and sluggish, very susceptible to fatigue; they cannot collect their thoughts and find it difficult to occupy themselves; are indifferent to their surroundings and let things go as they will. In emotional attitude they are sad and troubled, sometimes irritable, and occasionally at night they suddenly develop a state of great anxiety. In the severe types hallucinations of all the senses may be present, disorientation with confusion of thought, fantastic delusions and active excitement with anxiety. Emotionally, dejected and at times ill-humored, obstinate and resistive. Physically there is faulty nutrition and insomnia. It may be differentiated from dementia præcox by greater affect and disturbance of apprehension and orientation at the onset of the disease, and by the absence of negativism and stereotype movements; from the depressive phase of manic-depressive insanity by the absence of psychomotor retardation and the presence of faulty memory. The prognosis is unfavorable; after months of institution treatment, only one-half of the cases recover; the other cases improve gradually, but present as residuals weakness of intellect, poor judgment, inability to keep up with their classes, take up their former occupation, or provide for themselves.

A few cases are reported that seem to come quite properly under the heading exhaustion psychoses, as they apparently arise from excessive exhaustion or insufficient restoration of the nervous elements in the cerebral cortex. There is usually a history of a severe attack of scarlet fever, and evidence of a radical change of the physical organism. But even here one cannot always exclude the possibility of toxemia arising from the destruction of tissue.

Last, but not least, the relation of scarlet fever to dementia præcox must be considered, especially in view of the fact that so seldom can a definite or satisfactory cause for dementia præcox be ascertained. It is conceded, by good authorities, that, now and then, it follows very closely an attack of scarlet fever and bears a direct relationship to the same, but more often it is consecutive to some form of psychosis previously mentioned. It is the ***indirect*** relationship of scarlet fever to dementia præcox that suggests any ground for discussion. Years may have intervened be-

tween the attack of scarlet fever and the onset of the psychosis. Cases have come under our observation at the New Jersey State Hospital at Trenton with intervals ranging from 7 months to 31 years, with an unquestionable history of the fever indirectly influencing the psychosis.

The disease may have been forgotten or recalled only after careful and specific questioning; at the same time we find a history of convulsions, a delirium, physical weakness, otitis, loss of energy, long or final absence from school or arrested development. Too often the papers committing these cases to the psychopathic hospitals contain no statement whatever indicating the etiology of the psychosis; or such statements as overwork, mental strain, masturbation, intemperance, ill-health, cigarette smoking, a fever, feeblemindedness, etc. On the other hand the examining physician encounters much ignorance on the part of the parents regarding the diseases their children have had. Such statements as "a fever" or "a rising in the head" are only suggestive, not to mention the great number of aliens committed to our hospitals who are practically out of the question.

Kraepelin, in his study of the etiology of dementia præcox, has found that in 10 per cent of the cases there is a previous history of some severe acute illness, particularly typhoid or scarlet fever, from which time the patient has exhibited some change, as increased irritability, susceptibility to fatigue, or impairment of the full mental capacity. From the records of the Trenton State Hospital, we find a case corresponding identically with this view.

CASE No. 1.—F. C., male, white, single, age 24 years; occupation, laborer. Birth and infancy normal, strong and bright until six years of age, then had scarlet fever. Apparently good recovery, except mental dullness, more noticeable in his school work. Never independent of his parents, but earned wages. Admitted May 1, 1908. A year before admission a change in disposition occurred; he became seclusive and apprehensive; later suspicious of food, showed peculiar attitudes and mannerisms, was afraid to go to bed at night, had hallucinations of sight and hearing, at times untidy; disorientation and deterioration decided at the present time.

It will also be observed that the history of this case, prior to the onset of the psychosis, is practically the same as we find in those of arrested development or acquired imbecility, varying in degree rather than in symptoms. The following case illustrates this point.

CASE No. 2.—W. C., female, single, age 63 years. Family history negative. Personal history: Not unusual until eight years of age; then had an attack of scarlet fever with convulsions. Went to school very irregularly after this on account of her delicate physical condition. She could not keep up with her classes, became discouraged, lost interest in her studies and preferred to stay at home with her parents. After her first menstruation she became stronger in body, but her mental state remained impaired; never self-supporting, but a good helper in her own home. She continued on through life without any essential change except that now and then she had irritable and contrary spells. During the past year, symptoms of senile deterioration were observed. She would wander aimlessly about the house or away from home. Meddled with fire and matches, imagined she was going to starve, and had no clothing to wear; up and about her room at night, accumulating numerous and worthless articles. Committed to State Hospital October, 1908.

Again the question might arise, Why should the first case become one of precocious dementia, while the second continues a high-grade imbecile until arriving within the period of senile deterioration?

I might enumerate cases indefinitely, but that would simply add tedium without compensation. There is yet one condition that seems sufficiently important to justify a few additional remarks. As stated in the early part of this paper, otitis as a complication in scarlet fever has been recorded as high as 75 per cent in some epidemics, with deaf-mutism as high as 23.5 per cent, which makes a population surprisingly large and particularly susceptible to episodes which cause them to be sent to our hospitals.

CASE No. 3 illustrates this class. W. S. B., male, white, single, age 30 years; occupation, painter.

Born at full term, labor normal, fifth in line of birth, breast-fed; cut teeth, walked and talked at usual time. Strong and healthy child until about five years of age, when he had an attack of scarlet fever, discharge from both ears, lost sense of hearing, gradually lost his speech; later attended school for the deaf, learned the sign language and painter's trade, and was employed as carriage painter for four years in his home town; laid off because of slack work, became suspicious, irritable and threatening, homicidal and suicidal, carried razors and attempted arson; arrested and committed to the State Hospital. No deterioration. A prompt recovery.

In conclusion, first, allow me to repeat the words of Dr. Earle. "If insanity is to be diminished it must be by prevention and not by cure." Second, that scarlet fever is an etiological factor in the

psychoses and worthy of more consideration. Third, that it is more apt to be followed by complications and sequelæ and impair the future usefulness of the individual when it occurs during the evolution period; hence a most rigid quarantine should be instituted in each and every case of the disease and prophylaxis should be the "battle-cry."

THE GANSER SYMPTOM AND SYMPTOM-COMPLEX.*

REPORT OF FIVE CASES.

By THEO. I. TOWNSEND, M. D.,

First Assistant Physician, Dannemora State Hospital, Dannemora, N. Y.

Since attention in this country was called to the Ganser symptom by Dr. Adolf Meyer in the Pathological Institute for the New York State hospitals in 1904, Dr. A. H. Ruggles' article in the AMERICAN JOURNAL OF INSANITY of October, 1905,[1] and the able report of a case by Dr. Henry P. Frost at the 1906 meeting of this Association, published in the AMERICAN JOURNAL OF INSANITY of January, 1907,[2] an interested outlook for this symptom has been kept by the present reporter. It is undoubtedly an hysterical manifestation, and it is with some hesitation that five cases of this admittedly rare disorder are presented occurring in a year's admissions. In none of these, however, was it developed other than spontaneously, nor was there a question of malingering—even in the case of one patient who afterward claimed that he had feigned the symptom.

The typical Ganser symptom-complex is a confusional state, with hallucinations, of rapid onset and short duration; hysterical features are present, and the patient, in addition to silly answers and talk, misnames objects with analogous names, or describes their attributes in a roundabout manner; the replies to questions are often incorrect or absurd, but show a good understanding of the question and a near relation to the correct reply. To this latter symptom the term "Danebenreden" has been given by the Germans. Ganser states that on recovery there is amnesia for the attack.

The symptom of "Danebenreden," roundabout talk, or "symptom of approximate answers," as Dr. Ruggles has aptly termed it, is said to have been observed in dawn states on awakening from hypnosis, hysteria, manic conditions, melancholia, alcoholism, dementia præcox, imbecility, and to be most common in criminals and malingerers.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association at Atlantic City, N. J., June 1-4, 1909.

psychoses and worthy of more consideration. Third, that it is more apt to be followed by complications and sequelæ and impair the future usefulness of the individual when it occurs during the evolution period; hence a most rigid quarantine should be instituted in each and every case of the disease and prophylaxis should be the "battle-cry."

THE GANSER SYMPTOM AND SYMPTOM-COMPLEX.*

REPORT OF FIVE CASES.

By THEO. I. TOWNSEND, M. D.,

First Assistant Physician, Dannemora State Hospital, Dannemora, N. Y.

Since attention in this country was called to the Ganser symptom by Dr. Adolf Meyer in the Pathological Institute for the New York State hospitals in 1904, Dr. A. H. Ruggles' article in the AMERICAN JOURNAL OF INSANITY of October, 1905,[1] and the able report of a case by Dr. Henry P. Frost at the 1906 meeting of this Association, published in the AMERICAN JOURNAL OF INSANITY of January, 1907,[2] an interested outlook for this symptom has been kept by the present reporter. It is undoubtedly an hysterical manifestation, and it is with some hesitation that five cases of this admittedly rare disorder are presented occurring in a year's admissions. In none of these, however, was it developed other than spontaneously, nor was there a question of malingering—even in the case of one patient who afterward claimed that he had feigned the symptom.

The typical Ganser symptom-complex is a confusional state, with hallucinations, of rapid onset and short duration; hysterical features are present, and the patient, in addition to silly answers and talk, misnames objects with analogous names, or describes their attributes in a roundabout manner; the replies to questions are often incorrect or absurd, but show a good understanding of the question and a near relation to the correct reply. To this latter symptom the term "Danebenreden" has been given by the Germans. Ganser states that on recovery there is amnesia for the attack.

The symptom of "Danebenreden," roundabout talk, or "symptom of approximate answers," as Dr. Ruggles has aptly termed it, is said to have been observed in dawn states on awakening from hypnosis, hysteria, manic conditions, melancholia, alcoholism, dementia præcox, imbecility, and to be most common in criminals and malingerers.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association at Atlantic City, N. J., June 1-4, 1909.

CASE I.—M. N. Admitted March 15, 1907, from New York State Reformatory at Elmira; male; single; 18; nativity, New York State; father, Canadian; mother, New York State.

Family History.—Patient is the ninth of eleven children. A sister had epilepsy in childhood. On paternal side "there has been insanity, but very distant." Mother's family "nearly all have heart trouble."

Personal History.—"Typhoid pneumonia" at the age of seven; addicted to excessive cigarette smoking from an early age; attended school from six or seven to fourteen or fifteen years of age; his mother stated that he was bright in school, but his education was found to be very defective. After finishing school, he ran away from his home (on the St. Lawrence river) to Rochester, working in machine shops, and returned to work in a hotel. He had become addicted to the use of alcohol and had stolen before. One night he robbed a man in the hotel, was caught, convicted of burglary in the second degree and petit larceny, and sentenced to the N. Y. S. R. at Elmira with an indeterminate sentence of ten years maximum.

Psychosis.—The medical certificate from the Reformatory states that he had an attack of mania the previous month, and about ten days before admission became excited, screaming, spitting on the floor, making attempts to get out of bed, and was kept in a restraining sheet. He was uncleanly and filthy, attempted to eat his feces, urinated and defecated in bed, masturbated at every opportunity, and yelled and fought the nurses. His conversation was incoherent and he did not reply to questions. He said, "I can hear my mother's voice—I love it—I love it—there she goes—goes—goes—come here."

Physical Status.—A fairly well developed boy of medium height, with stigmata of degeneracy; high V-shaped palate; irregularly implanted teeth; head flat posteriorly; poorly nourished with slender musculature; hæmoglobin, 85 per cent; recent vaccination and circumcision both unhealed, probably accounting for a temperature on admission of 100.6; pulse, 100; respiration, normal; this rise of temperature subsided in four days. The subjective sensations were of depression and confusion, and a feeling of his eyes changing—"they feel queer and I can't see well; it seems as though they were crossing." There was an insufficiency of the right external rectus muscle, causing a frequently recurring strabismus to account for this feeling. Pupils normal, and no limitation of visual fields; twitchings of the left lev. lab. sup. alæquenasi were also observed; patellar reflexes markedly exaggerated—Achilles slightly; fine tremor of tongue, face and hands; slight bronchitis; appetite poor; tongue coated; mouth covered with sordes; breath fetid; constipation with scybalous stools.

Mental Status.—Restlessness; distractibility; suggestibility with a semi-delirium and the Ganser symptom-complex were manifested for four days after admission.

He was carried into the hospital, being very weak. His spontaneous production was very rambling, but gave some evidence

of flight of ideas, as follows: "You have started me on the road to hell—You started from a rattler—You started from a snake—You are a rattlesnake—Go in and get on the glass table" (evidently referring to his circumcision at the Reformatory), and his language became very vile, showing coprolalia without apparent appreciation of its meaning. What is your name? "My name is Carlisle" (more obscenity). "Give me that sewing machine" (no similar object in sight). "My father had a sewing machine at Fishers Landing, and I had a bicycle and fell off and shaved off my fingers in the spokes." Here he leaned forward and touched the attendant's uniform coat button with his tongue. He was not oriented for time, place, or person. His mood was that of anxious depression and he frequently spoke about cutting his head off. The following shows the Ganser symptom of "Danebenreden." Various objects were called to his attention and he was asked to name them. He would reach for everything shown, saying, "Give it to me," before replying. Pen knife? "That's a jack knife; that's a razor, shave my head off" (motioned as if to cut his throat). A pair of spectacles? "That's—A man can see through it with both eyes" (making circles with his fingers and holding them up to his eyes, imitating the appearance of a person with spectacles). Cap? "He pulls it right off his head" (imitating bowing with a cap). Electric light? "That's a curer of blue lights, let me get up and turn it on; give me that razor strops—true." Chair? "Mop-handling Jew, let me get up and I will go over and sit down on it." Cigarette? "A sneakingrette, a smoker of liars, a smokerette." Book? "Dictionary; give it to me and I'll pick 'em all out!" Key chain? "Jail man's chain; chain down my head and give me a hypodermic." Watch? "That's a timepiece; you start from the Grand Central Station and—" Match? "That's a smoker—a piece of—give it to me and I'll light it." Pencil? "That's a stinker of lies; give it to me and I'll show you what to do with it." He took it and made a motion to write, but when given a piece of paper and asked to write, made a vulgar picture of female genitalia, and imitated the motion of masturbation. Bunch of keys (rattled before him)? "That's a rattler of stinking snakes." Dollar bill? "That's one hot ball." The following day called it "one green." Bottle? "That's a killer of lies; that's a kidney cure." Newspaper?

"That's to read." The word opium was suggested, and he said, "One opium—hop joint—hop box—hop chewer—curer of lies." How many are there in your family? "There are a thousand in our family; you will know me a thousand years from now." How old are you? "I am a thousand years old." (He imitated several somewhat complicated motions of the examiner's hands.) The following day named objects as follows: Empty cartridge? "Shoot me dead; shoot my head off." Pair of shoes? "Shine them everywhere you go." Tooth-pick (snapped to attract his attention)? "That's a snapper to pick all my teeth with." Knife? "Cut my head off." Why do you want me to cut your head off? "Because I am the dirtiest man in the world." Lead whistle? "Blow it." Fifty-cent piece (with the eagle towards him)? "Eagle." Match? "Smoke it." He continued to talk a great deal about "rattlesnakes" and "a curer of lies." Four days after admission he began to be a little clearer, saying, "Doctor, bring me back to my senses again, I am getting clearer every day." He still continued to talk about rattlesnakes, cutting his head off, etc., and said, spontaneously, "I know you" (beginning to cry). "I stole your two dogs; poor boy, chop himself all to pieces." He did not know how long he had been here, and could not recall names, but said that his birthday was on June 6, and that he was 18 years old. He did not show the Ganser symptom at all after this, and the mental status was completed on the fifth day, showing defective orientation, poor memory for the remote and immediate past, and defective retention, with limited grasp on education. Calculation showed mistakes and slowness with fatigue at the end of the examination. He read a short newspaper article fairly well, but elaborated slightly in subsequent rendering. His insight was partial. On admission he once said, in his rambling talk, "I'm crazy over a woman." On the fourth day said that his head was clearer, and asked to be brought back to his senses; on the fifth day said that he was not quite all right and continued, "I never can be all right again." During the first two days he soiled and wet himself with indifference. Two weeks after admission was in a peculiar dream-like state, in which he kept his eyes closed, made slow gestures with his hands, and muttered almost continuously, but slowly. He held his hands out straight before him and said, "Oh, that gold

tooth, and that dentist's chair" (unintelligible mutter); "and I met an old gray-whiskered man." When spoken to sharply he opened his eyes and said, "Yes, sir." Did you sleep last night (sleep defective)? "No, sir, I started to sleep, and I began to dream the same old dream. I confess the whole business; a man lay there with three fingers held up and a monkey nose" (closed his eyes and began to mutter again). Why do you keep your eyes closed? "I can't read that dream with my eyes open." (The telephone bell rang.) "There, do you hear that bell? There's that straight jacket again, that horse blanket." (Humming.) "'The frost is on the pumpkin, the pumpkin's on the vine.'" Spoken to sharply, he again opened his eyes, looked intently at the examiner and commenced to cry. "You're my mother, I know it, you're my mother, aren't you?" What makes you say that? "Because you've got my mother's eyes." He stated that he heard his mother when he went to sleep, and that he could hear her in spirit any time he wished. "I don't know that it's a real voice, but it is the spirit of a voice. I want to go to sleep and never wake up." The following day he again partially cleared up and was quiet, but two days after this was depressed, and said, "I am going to be chopped up to-day or shot to pieces, ain't I?" Why? "Because I'm nobody, I tell so many lies," beginning to cry. He was easily reassured though, and said, "Oh, I am so thankful." A month after admission it was noted that he was less emotional, but he said, "Thoughts keep coming into my head annoying my mind; seems as if my brother Al and poor mother was here, then that she was dead. I know I done some awful wrong, but was in awful pain when I come in here, and would have been glad to have my head cut off, but I don't think so much about it now." Vaccination and circumcision had completely healed, and he had improved physically. The following day after this note he was elated, laughing considerably, or talking and singing in a religious strain all day. When asked why he laughed, replied, "Oh, I'm so happy." A little too happy, are you not? "Yes, I'm 'most too happy in the love of Jesus." Soon after this he had double parotitis and was in bed for three days, and then depressed and confused, with his mind running again on thoughts of being killed. This condition lasted for two days, and after that his improvement was continuous. He worked

satisfactorily and well in the shoe shop, and became quite expert in shoemaking. He was rather quiet and not inclined to voluntarily converse, but was polite, willing and respectful. His insight as to his former condition was good, but memory of the attack was slightly hazy, and he was disinclined to discuss it. He was discharged recovered, and returned to the Reformatory.

CASE II.—C. J. C. Admitted Aug. 27, 1907, from Sing Sing Prison; male; single; 24; nativity and parentage, Italian.

Family History.—Obtained from patient, who stated that he was the elder of two children, but that after his father's death his mother had married again and there were now seven other children in the family. He did not know of any insanity in the family, or the cause of his father's death.

Personal History.—Patient came to the United States at the age of 13 and worked about as a bootblack in New York City, in a coal cellar, etc., in the intervals going to school, but played "hookey" a great deal. For eight years prior to his crime he worked in a piano factory. He became intemperate, got into bad company, and "we broke open a store. I was only the accomplice; my friend took a plea and got ten months, but I went to trial and got one to three years." He was sentenced to Sing Sing Oct. 7, 1904, for burglary in the third degree with the sentence as stated.

Psychosis.—The certificate of the prison physician states that about a week before admission he began to talk incoherently of horses, laughed, shouted and kept this up almost night and day, pulled off his clothes, and they were obliged to use restraint. It was further stated that he had been "an all around bad man and punished very often."

Physical Status.—A fairly well built man of medium height; head large but symmetrical; stigmata of high palate with central torus; ears small in proportion to size of head, and left ear flares more than right; hæmoglobin, 90 per cent; his body was covered with old scars, most of them received in a fight in the prison. There was nothing of importance in the rest of the physical examination, except that he could not differentiate the test odors. There was a tendency to constipation.

Mental Status.—Restless excitement on admission with silly talk; flight of ideas; Ganser symptom; violent resistiveness with clouded sensorium; Ganser symptom only elicited on admission.

On admission the patient was restless, excited and elated, laughing often. His answers to questions were silly, as a rule, and sometimes showed the Ganser sign of "Danebenreden." When asked his name, replied, "Paul, Pigeon, Carl, Frenchy." Occupation? "Putting salt on pigeons to make them run." Age? "Since I have known you I don't know my right age." Where did you come from? "From the ocean, I am just off the boat." What

day is it? "I don't sail it." Question repeated. "Monday (Tuesday) August, 1907." Objects were called to his attention, and he was asked to name them with the following result: Pencil? Patient made a motion of writing, without answering. Question repeated. "That's a pen." What would you do with it? "Paint with it." Why do you say that? "Because that helped to make a bull dog." Knife? "Knife." Match? "To burn." Watch? "To steal." Question repeated. "Watch." What is the trouble with you? "I just fell overboard—I found a golden egg in the inn; I didn't find it, Paddy found it; I looked for Paddy, and Paddy looked for me." He made several attempts to attack the attendants on the night of admission, and was found to have a slung shot in his possession, made of a strip of sheet and a large piece of mortar. In two days he had cleared up considerably so that he was oriented and talked rationally, although still slightly elated and inclined to laugh without sufficient cause. He then revealed a persecutory trend connected with his prison life, and said, "I didn't know what I was talking about when I came in; I guess I was off my head." His memory of the remote and recent past was good; retention defective; grasp on education fair; calculation quick and correct. He explained that he did not intend to use the slung shot as a weapon, but that he thought the door had some kind of a patent arrangement, and that if he threw this over it, it would open; and that his fights with the attendants were due to his imagination that the medicine was poisoned. He improved continuously from this time, but when discharged after 41 days, on the expiration of his sentence, he was still emotional, being easily moved to tears or laughter.

Case III.—N. C. Admitted Nov. 26, 1907, from the New York State Reformatory at Elmira; male; single; nativity and parentage, Italian.

Family History.—Obtained from patient. He was the youngest of 12 or 14 children; all the others died in infancy but one sister who is now living and well. He knew of no insanity in the family.

Personal History.—Patient came to the United States when 3 or 4 years old, soon afterward going to New York City. He attended school from the age of 10 or 12 to about 16. After leaving school he gives a history of having worked in eight different places in grocery stores, factories, laundries, and once as a conductor on a street railway in Mt. Vernon, but the longest steady employment which he had was for five or six months. He admitted thieving and getting into fights as the reason for terminating

his service in most of these places. The history of his crime as given by himself is as follows:

"I came home one morning and found a girl about 8 or 10 years old in the house. I asked my sister who she was, and she said she didn't know, but that she found her wandering around alone and took her in. I said that a lot of Italians got into trouble from this, and that she had better tell the police and not keep her; so she went out, and the little girl started to run away, and I chased her back in the house and she went and hid under the bed. I didn't do nothing to her. I'm a Brotherhood of St. Andrew. They arrested me and charged me with assault and rape, and the jury disagreed. On the second trial the Children's Society said that they would teach the girl how to answer so as to give me 20 years for rape, and my lawyer and minister advised me to take a plea of assault; but I'm innocent." He was sentenced Nov. 13, 1907, for assault in the second degree (confession) with a term of five years maximum.

Psychosis.—The medical certificate states that about five days before admission he suddenly became excited, laughing and yelling without reason; talked loudly and incoherently; sat down and got up repeatedly; attempted suicide by hanging, and it was necessary to keep him in restraint. In the presence of the examiner he said, "Gentlemen of the jury, this man is innocent—Bring me my fancy pants! Correct! Stand up! Correct! Sit down! I am King Edward, the big gun of the whole outfit."

Physical Status.—A fairly well developed but undersized (5 ft. 5¼ in.) Italian boy with stigmata of retreating forehead and chin; ears small and close to head; slight Darwinian tubercle on left ear; right eye enucleated; wears artificial eye; teeth uneven; palate shows posterior central torus; good nutrition; hæmoglobin, 90 per cent; special senses acute and normal; patellar reflexes slightly increased; slight tremor of tongue and hands; sleep restless, with almost nightly libidinous dreams and emissions; gonorrhœa four years ago with subsequent attack about five months ago.

Mental Status.—Restless excitement; attempt at suicide; confusion; foolish talk; mood elated, laughing without cause; distractibility; absurdly wrong answers to questions.

On admission patient never answered any questions correctly, saying he was of Scotch birth; had two fathers; was 32 years old; was born in Jamaica by the City Hall; had been in Elmira three years (two weeks). He said, spontaneously, "They say I'm crazy; I ain't bug house—I didn't kill anybody; I didn't get sentenced to Elmira. I am Rockefeller; am I going home now or when am I going? I didn't kill anybody." When asked the date, replied, "I don't know—Winter time—January." He said this was the Dannemora Prison, and the examiner the president's son. Laughed without cause, and was distractible. The following day he was smiling and alert and answered questions as to his

pedigree correctly, but professes not to remember that he said anything unusual on admission, and denies that he has ever been insane. He was not exactly oriented, giving the date as October 25, 1906 (November 27, 1907). Five days after admission he admitted that he remembered having given foolish answers to questions at first, although prior to this he had continued to assert that he did not remember anything about it. He claims that he did it on the advice of a runner in the jail at Plattsburgh, where he spent the night on the way here. He affirms that this man told him that if he talked and acted foolishly he would soon get out, but he did not seem to appreciate the illogical nature of this. He was well oriented, but continued simple and defective, smiling in a foolish manner when addressed, and was unable to employ himself satisfactorily in any situation in the hospital. He continued to assert that he feigned insanity on admission, but gave no evidence of delusions or hallucinations at any time, and after 14 months was discharged recovered by return to the Reformatory. I have no doubt that if this patient had been asked to name objects, he would have exhibited the Ganser symptom more fully, as he was in a confused state very similar to the others; but unfortunately the test was not applied.

Case IV.—J. W. Admitted Jan. 21, 1908, from the New York State Reformatory at Elmira; male; single; 21; nativity and parentage, U. S.

Family History.—Obtained from patient. He was the oldest of four children, the others living and well. Father and mother both born in New York City; the father was a horse dealer, and died about three years ago of heart trouble. He was intemperate at times.

Personal History.—Obtained from patient. He was born in New York City Jan. 26, 1887; he knew of no illnesses. At the age of 9 or 10 he fell on his head from a swing and struck on a stone. There is now a depression in the frontal bone at the hair line. Began school at the age of eight or nine, attending three or four schools until the age of sixteen. He played hookey a great deal—several times a week. Began smoking cigarettes to excess at the age of thirteen; began to drink to excess at sixteen, becoming drunk three or four times a week. At this time he commenced working as a driver for a butcher, but after one year left for higher wages as an elevator boy in an apartment house for three or four months; was discharged for being late to work. He then learned bricklaying at a trade school, and worked for one year; then got into bad company, and at eighteen, with three other boys, burglarized a house of jewelry. He was caught in the house and sent to the House of Refuge for one year. He had a fight there and was sent to the Eastern New York Reformatory at

Napanoch for two years. After his release he worked at bricklaying six or seven months. He then assaulted a man by striking him on the head with a stone while drunk. He was sent to Elmira for five years maximum. He was paroled after eighteen months. He worked for eight months as a tinsmith with an uncle, but then left New York and went to Oneida with two others to burglarize a store; they could not accomplish it however, and broke into a railroad station, stealing $10.00 or $15.00 and some jewelry from baggage; were caught in Norwich, and sentenced for a term of five years maximum for burglary in the third degree (confession); one of these boys (A. J.) is now a patient here.

Psychosis.—The medical certificate from the Reformatory states that about nine weeks before admission the patient became violently excited. He would not sit quiet, made frequent passes at imaginary persons, made motions as of scalping his enemies, shooting them (with loud bangs), refused to work, made frequent assaults, and required to be restrained. It was also stated that he had had attacks of acute mania which subsided. He said in the presence of the examiner—"Oh, if I had a gun! I'll do life and then I'll do a day—If I could only cross swords with Major Chatfield I would run him clean through. Bring me two orders of chocolate soup."

Physical Status.—A fairly well developed boy, but undersized (5 ft. 5¼ in.). Forehead low and retreating; palate high and very narrow; teeth in poor condition and irregularly implanted, seven missing; general nutrition good; hæmoglobin, 95 per cent; tattoo marks of a spread eagle with the American flag on the right forearm; pupils dilated, and reaction to light and distance sluggish. Taste.—Did not distinguish salt, sweet, sour or bitter. Smell.—Did not recognize any difference between camphor, wintergreen, peppermint, cloves or lemon. Cutaneous sensibility normal for localization, heat and cold; but for pain absent. He said a pin was a "tickler," and he would push it into his flesh and say it felt good and did not hurt him at all. Patellar reflexes much diminished; fine tremor of hands and tongue; sleep defective; stated that he dreamed about horses, automobiles and airships. Constipation.

Mental Status.—Disoriented; elated; delusions of persecution and grandeur; restlessness; Ganser symptom lasting three days. On the way to the hospital he was elated, thought he was sailing along in an airship, and laughed a great deal without reason.

On admission the patient was disoriented as to time and place; said that he did not know his first name—that his last name was Wallace. He stared at things about him and answered almost no questions. Said that he did not know where he came from, and when asked his age, replied, "I don't know the last time I saw him." He was confused and could not tell the name of common objects, but would look at them intently without making any reply. When a pocket knife was shown him he appeared terrified

and made a lunge for it. The day after admission he was a little more talkative, and while he would not name objects would refer to their use as follows: Pencil? "What you scribble with." Match? "Wood—he make fire with." Necktie? "Put it around your neck when you want to feel good." Buttonhole? "Hold the button." Shirt? "When you go to see a Molly." Tumbler? "To feed your tickler." Sponge? "Why, if you want to wet your ear." Bottle? "Bottle." Nail brush? "Take that for me teeth." Electric light? "That's a light; when you go to sleep the light goes out." Soap? "Piece of soap." Watch? "Timepiece." Money? "Get something for me." A dime? "That's a big one." Nickel? "That's a small one." Handkerchief? "Rag." Book? "I don't know—you got me on him." A postage stamp? "That's what you put on him" (pointing to an envelope on the table). Toothpick? "Piece of wood to chew." Pencil? "Scribbler." At this time the patient was smiling and seemed to want to talk, and laughed a great deal without reason. He said that he wanted something to eat to make his "tickler feel good"; by "tickler" he said he meant something in the region of his stomach, which he referred to as "he"; said he did not care about eating, but ate to make his "tickler feel good." On the third day he said that he had lost all his airships. Asked again about his father's and mother's names, which he refused to give on admission, said "I don't want to know, that's all."

He stated that he "Would like to have a great, long sword, that would grow longer and longer, and give Major Chatfield a short one and march him about. I would like to cross swords with the major and make him walk around the institution and get all the other inmates to follow, and get them to make fun of him, and get them all crazy." This was because the major had put him in the guard house, and he would like to get square with him. The patient here laughed loudly, as if he had heard a good joke. He said that while in the guard house in the Reformatory he felt confused, and was unable to make himself do the work that was required of him. When asked about his laughing, said, "I can't help it, I must laugh, I always did laugh." He said that he "Would just as leave stay here for all time, if necessary." He thought himself a very smart boy, and a good fencer and boxer. He answered questions rather slowly, his eyes were shifting, he

rocked from side to side in his chair, and constantly picked at his sleeve or at his pockets. He thought he had been here three or four nights (two nights), but recalled the menu of recent meals correctly. Retention was very defective, and his education limited. His calculation was slow and defective, but there was no subjective difficulty; he did not appreciate discrepancies. He had no insight into his condition, saying, on the third day, that he was all right, but that he had been driven crazy in the Reformatory. Later, on the third day, when the correct names of objects were insisted upon, he would name them, and appeared much brighter. He would give incorrect answers at first as follows: Pencil? "A scribbler." What do you do with it? "You scribble with it." When the correct answer was insisted on, he replied, "Well, you can call it a pencil if you like." He gave similar replies when asked to name money and a watch. After this the Ganser symptom could not be elicited. He improved physically and began to employ himself industriously, and after a month was transferred to the working ward. He was rather quiet and seldom spoke to the other patients, and soon afterwards developed periods of sullen depression, lasting several days, when he would refuse to speak to the physician, or become tearful. Once he refused to eat for several days, and finally admitted that he thought it was because something was put into his food by the cook. He showed himself very simple minded, and associated with malcontents on the ward. At present (April 20, 1909) he is rather quiet, but sullen. He recalls his foolish answers to questions on admission. He states that he does not know why he should answer so, but that his mind was mixed up, and that he said the first thing that came into his head.

Case V.—F. G. Admitted March 3, 1908, from Auburn Prison; male; nativity, Holland; age, 28; single; painter. Anamnesis not obtained. He was sentenced for burglary in the third degree (confession) from New York City for a term of five years. His prison record is as follows:

One term, workhouse, June, 1898, for five days, for intoxication.

One term, State Prison, for three months, for disorderly conduct.

One term, Sing Sing Prison, as H. DeW., May 31, 1899, for four years and six months, grand larceny first degree.

Psychosis.—The medical certificate from the prison stated that about a month prior to admission he became noisy, yelling and screaming at night. In the presence of the examiner he begged to get out to work. He lay in bed naked, and would not get up. It is also stated that he was destructive.

Physical Status.—A fairly well developed man, but undersized (5 ft. 5¼ in.); stigmata of low forehead; receding chin; nose hooked, with deviation to the left; palate high, with posterior central torus; nutrition poor, and musculature soft and flabby; hæmoglobin, 85 per cent; mucous membranes pale; pupils normal; hearing slightly defective in left ear (a piece of pearl button was found in this ear and removed with improvement in hearing); responses to the tests for taste and smell were foolish; cutaneous sensibility normal; reflexes exaggerated; fine tremor of tongue and hands; small area of consolidation in left apex above the third rib; appetite poor; tongue coated white; bowels constipated.

Mental Status.—Delirium with Ganser symptom-complex; restlessness; distractibility; delusions that he was a gorilla; foolish talk; objects misnamed.

On admission he was in a restless delirium. He said, spontaneously, "I want to know what I am signing; I came here for my wife and four children; do you give them up?" When asked to show his tongue, he refused, and said, "I show no tongue; you know who I am" (observing the clock); "that clock will never tick the same hour again, you know." What date is this? "March 1, 1908." What is the day of the week? "The first day of the week." What day is that? "Damned if I know; do you?" What place is this? "This is the Women's Prison at Dannemora, built on the harem plan; it was built by my father and mother, they are two genuine gorillas." Who is this (pointing to an attendant)? "That's Johnny Cully, my deputy, I'll make him—" (vile obscenity). "I claim this place—the gag is up—the jig is up—you had better throw up the sponge." What is your name? "Henry Rinaldiny; I'm a full-blooded gorilla." Various objects were held up to him, and he was asked to name them with the following result: Pencil? "That's a thermometer." What would you do with it? "I'd stick it up my tupes; do you know what that is? I carry a dictionary in my pocket and in my tonsils." Watch? "An alarm clock." Pocketbook? "Letter." Five dollar bill? "Confederate money." Postage stamp? "Chinese stamps." Matches? "Elfelfas; elsulphurs." What would you do with one? "Light a match with it, of course." Rubber band? "Ruptures." Pen and pen holder? "That's a branch of a tree grasped by a gorilla; when you touch that you are impressed, you know." Two days after admission, after a good night's sleep, he appeared a good deal brighter and did not manifest the Ganser

symptom, and stated that he did not know what was the matter with him on admission, but the following day had relapsed into the same state. In the physical examination he said that salt was "The juice of julass." Sweet? "Juice of a gorilla." Vinegar? "Between sweet and sour." Quinine? "Taken from the heart of a gorilla." In testing his sense of smell he said that lemon was "Snarita—the juice of snails." Camphor? "Tulita—taken from my tooth." Cloves? "Taken from the skin of one of my dear children." Peppermint? "Juice of apple worms." Wintergreen? "Syrup of maple." Orange? "Gekerita, or fish juice." Asked how he slept, replied, "I sleep good, quite obstinickly." Six days after admission it was noted that he continued restless and silly in conversation. In reply to questions, said, "Oh, yes, I'm a gorilla all right; me mudder and fadder is in here, and I got a wife in here, too. I think she is over there where those icicles are" (pointing to the roof of the assembly hall). "She's run this institution for some time." He said the date was March 8 (9); that the hospital was "Elmira, or Dannemora Prison, or the bug house; everybody making funny signs and walking around and gravivating; they say they own the earth." When were you born? "Darned if I know." Where did you live? "I lived around anywheres, anywhere I would hang up my hat, 25th, 26th, or 27th Street." He recalled the menu of recent meals correctly, and said that he had been here one week (correct), and recalled perfectly the incidents on admission. He forgot the examiner's name in one minute; read a short newspaper article over very fast, and afterwards could not recall any of it, but fabricated, "They tore some bodies to pieces." Who discovered America? "I did," laughing. But, when pressed for an answer, replied correctly. What was the date? "Some years ago, a thousand years, I guess." His calculation was defective, but partly because of indifference. He was rapid in counting; "$9 \times 9 = 99$," and "$12 \times 13 = 139$," after giving several other sums correctly. When told he was wrong, said, "Well, figure it up yourself." During this examination he commented on the examiner's watch and scarf pin, and attempted to take them to look at. When reproved, he said, "You must not mind me, I'm a little childish; I want to touch things. I got a terrible pain in this hand. I had a fight with a fellow, I guess—No, it was a lion—Yes, I fought

with that lion for seven hours over in Paris—I tore his mouth open and ripped him up—You see, I fight with a clear conscience." When asked about his crime, said, "It was burglary; that's a cowardly crime, but I was hungry and needed the money." His handwriting showed tremor. He wrote his name as, "Henry Rinaldiny; prison name, F. G., No. 28,407" (correct). Up in the corner of the paper he drew a peculiar diagram, which he said was a secret emblem of his family.  Twenty days after admission he no longer misnamed objects, but continued restless, incoherent, and to assert that he was a full-blooded gorilla; he frequently smeared the walls of his room with feces; picked pieces out of the flooring of his room, and had an unprovoked fight with another patient. His tubercular lesion advanced rapidly, he lost in weight and developed a considerable pyrexia, his temperature never falling below 100°, and varying between this and 103°. Seven months after admission, his term of sentence having expired, he was recommitted as insane, but three weeks after this was taken home by his sister, presumably to die. Nothing has since been heard of the case.

Henneberg, in 1904,[1] claimed that the condition described by Ganser was very rare, not entitled to a distinctive classification, and goes on to observe that the symptom of "Danebenreden" may be found in normal persons when perplexed, and also when they intentionally give foolish answers to foolish questions. With this position Dr. Ruggles seems to agree, partially, at least, as the following quotation would show: "It seems fair to say that in many cases too much importance has been attached to 'Danebenreden,' and that further analysis would show that it was not a necessarily significant symptom, but a matter of suggestion, perversity, obstinacy, or carelessness. At least, for the dementia præcox patient, these conclusions probably hold good.

"It is possible that the symptom may have another meaning with hystericals, but this seems improbable when we consider the notorious perversity of these patients.

"The desire to answer incorrectly, combined with the wish to answer with the greatest possible ease, would seem to form a sufficient explanation for the larger part of the cases."

This is the conclusion of Dr. Ruggles' article, and may be true of cases which present the Ganser symptom alone, and are not in the confusional state. I have not, however, observed the symptom or the peculiar confusion before in the four State hospitals where my experience has been gained, and do not feel justified in drawing positive conclusions as to the distinction between the symptom and the symptom-complex.

In none of these five cases was a complete anamnesis obtainable, and the history of the onset particularly was incomplete. They were all felons and ranged in age from 18 to 28. The cause of insanity was not assigned in any case, but in Case I, with a diagnosis of manic-depressive insanity, mixed type, it was, at least, partly due to a recent circumcision and vaccination; Case II, diagnosis, manic-depressive insanity, manic type, had been punished very frequently in prison; Case III, diagnosis, manic-depressive insanity, manic type, made a false claim of malingering; Case IV, diagnosis, excitement not sufficiently distinguished, had a depression in the frontal bone from a fall 11 years previously, or at the age of 9 or 10; Case V, diagnosis, excitement not sufficiently distinguished, had advanced pulmonary tuberculosis. Four of the patients had been alcoholics, but over 72 per cent of the admissions of the Dannemora State Hospital are intemperate.

The two who were discharged recovered, the one who was discharged improved, and the one remaining in the hospital improved, did not have complete amnesia for the attack, but recalled, although not clearly, their answers which manifested "Danebenreden," and the cases were otherwise typical. No satisfactory explanation of this fact has occurred to me, but they were very closely questioned regarding their memory of the confused period. In all, the symptom was of short duration and sudden cessation. The fifth case showed the typical symptom for 20 days, but continued to give silly answers, and the confused state persisted until his discharge after seven months.

All appeared to be constitutionally inferior, and two had previously served terms for felonies. Constitutional inferiority is the rule among criminals, and explains why a frank case of manic-depressive insanity among the psychoses with which they are afflicted is a rarity, the great majority having a marked and somewhat similar paranoid trend.

With perverse and silly answers to the examiner's questions, in various widely differing psychoses, we must all be familiar; but the peculiar confusion and misnaming of objects seen in these cases and in the case reported by Dr. Frost seems to present a definite picture, and to merit the retention of the distinctive term in honor of the original observer, and to deserve the interest of further study.

BIBLIOGRAPHY.

1. R. Henneberg: Ueber das Ganser'sche Symptom. Allgemeine Zeitschrift für Psychologie, 1904, p. 621.
2. A. H. Ruggles: Observations on Ganser's Symptom. American Journal of Insanity, October, 1905, p. 307 (with a very complete index of references).
3. H. P. Frost: Hysterical Insanity: Report of a case presenting Ganser's symptom-complex. American Journal of Insanity, January, 1907, p. 301.

AN ANATOMICAL ANALYSIS OF SEVENTY CASES OF SENILE DEMENTIA.*

By C. G. McGAFFIN, M. D., Taunton, Mass.

The following analysis of the Taunton State Hospital autopsies on cases of senile dementia or cases so diagnosed by members of the staff of that hospital, was conducted independently of Dr. Southard's analysis of the Danvers State Hospital material, and subsequent to it, to discover whether the Taunton material, diagnosed by a different staff, would contain a similar grouping of conditions.

Clinics have been held similarly to the Danvers method, already described, but not all of these cases here considered have been passed upon by the whole or a majority of the staff, but by the superintendent and one assistant physician. Many, however, were presented at the staff meetings, and all the data that could be secured from that source have been used.

From the opening of the Taunton laboratory in May, 1898, to the first of January, 1909, 328 autopsies have been made. On 75 of these a diagnosis of senile dementia was made, or 23 per cent.

This high percentage is not unusual, and a similar one is to be found in the records of nearly every insane hospital and asylum in the country.

In five of these cases the head was not opened, leaving 70 cases as the basis for this report.

For many years it has been thought by psychiatrists that senile dementia was rather a "dumping ground" into which to cast many heterogeneous cases arising after the sixtieth year of life and showing symptoms, one or some of which might fall into the group of symptoms usually characterizing this psychosis. Too little attention was paid to arteriosclerosis of the brain as an entity, and it was held that such atheromatous change and senile dementia were identical and the one affecting the brain necessarily entailed a diagnosis of the other; also organic brain disease was too often overlooked.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

AN ANATOMICAL ANALYSIS OF SEVENTY CASES OF SENILE DEMENTIA.*

By C. G. McGAFFIN, M. D., Taunton, Mass.

The following analysis of the Taunton State Hospital autopsies on cases of senile dementia or cases so diagnosed by members of the staff of that hospital, was conducted independently of Dr. Southard's analysis of the Danvers State Hospital material, and subsequent to it, to discover whether the Taunton material, diagnosed by a different staff, would contain a similar grouping of conditions.

Clinics have been held similarly to the Danvers method, already described, but not all of these cases here considered have been passed upon by the whole or a majority of the staff, but by the superintendent and one assistant physician. Many, however, were presented at the staff meetings, and all the data that could be secured from that source have been used.

From the opening of the Taunton laboratory in May, 1898, to the first of January, 1909, 328 autopsies have been made. On 75 of these a diagnosis of senile dementia was made, or 23 per cent.

This high percentage is not unusual, and a similar one is to be found in the records of nearly every insane hospital and asylum in the country.

In five of these cases the head was not opened, leaving 70 cases as the basis for this report.

For many years it has been thought by psychiatrists that senile dementia was rather a "dumping ground" into which to cast many heterogeneous cases arising after the sixtieth year of life and showing symptoms, one or some of which might fall into the group of symptoms usually characterizing this psychosis. Too little attention was paid to arteriosclerosis of the brain as an entity, and it was held that such atheromatous change and senile dementia were identical and the one affecting the brain necessarily entailed a diagnosis of the other; also organic brain disease was too often overlooked.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

Needless to say, much of this doubt and obscurity has been cleared away in the past few years, but five to ten years ago this was not the case, and the diagnoses made up to 1904 are probably much garbled by this old idea.

Noetzli, in an analysis of 70 cases of senile dementia, said that atheroma of the cerebral blood-vessels was the fundamental cause of senile dementia, and that the atrophy of the brain came as a result of this atheroma; also that the frontal lobes were not more atrophic than other portions of the cortex. Some of the figures obtained by this analysis do not seem to coincide with those ideas.

Of the 70 cases considered in this paper, in eight there was some doubt expressed by some members of the staff as to the diagnosis; and these are set one side, leaving 62 clinically uncomplicated senile dements. In 56 cases some note was made regarding macroscopic atrophy of the brain and of this number 26 showed a more marked atrophy of the frontal convolutions than in other parts of the cortex, or 46 per cent, and in only five cases was general atrophy observed.

This would seem to point to the conclusion that the frontal convolutions are attacked first or that they withstand the atrophic process least well. Again the higher centers of thought, judgment and reasoning are the points hit first and hardest.

General atrophy is not common and the usual procedure is for certain parts of the cortex to be picked out for local atrophy. General atrophy in itself would point toward a condition of idiocy, but it would be difficult for anyone to call senile dements idiots.

Focal or local atrophy then is the usual finding with the frontal convolutions the focus of location.

In 44 cases of the original 62 the brain weight was given and of these 27 were males and 17 females. In handling these figures of weight the accepted averages have been used: 1358 gm. for male brains, and 1235 gm. for females.

Of the 27 male brain weights 17 showed to be below the average, or 63 per cent. Of the 17 female brain weights, on the other hand, 14 were below 1235 gm., or 82 per cent.

Of the whole number, seven showed a loss of one-sixth in weight and may be considered truly atrophic; of these two were males and five females. Of these seven atrophic brains by microscopic examination, six showed distinct loss of cells in the layers

of the cortex, especially the outer layer, and a general thinning of the cortical mantle.

From these figures it would seem that the female brain tends to lose in weight under the action of senile changes more commonly than males and that the loss is greater. The female brain is normally 123 gm. lighter than the male, and so the loss is correspondingly even greater in extent.

From the clinical records the duration of the psychoses and the age at death has been studied, in those cases whose brains at autopsy showed some loss of weight, and the averages obtained.

In the 17 males, the average duration of the disease was four years, eight months and six days, and the age at death 75.

In the 14 females, the duration was four years, one month and 13 days, and the age at death 79 and six-fourteenths.

The figures here for the duration do not exactly coincide with those given by Southard and Mitchell last year, but that is not surprising as they were not considering senile dementia, but in relation to the average age there is a striking similarity.

The age at onset in these cases is interesting, as it will be seen that the males showed senile deterioration about four years before the females. Perhaps that is a direct result of the more strenuous life men are supposed to lead and that they are more prone to arterial changes for the same reason, but these men had enough vitality to prolong their hospital existence seven months and 23 days longer than the women.

Was it because they could take care of themselves better among the other patients than the women? Do our male attendants excel the female in efficiency? or, Was it the added four years to their lives that made the women the easier prey of terminal disease?

With regards to arteriosclerosis, in 11 cases it could be fairly considered from the macroscopic description that there was sclerosis of the cerebral end arteries. Spots of focal softening mentioned in the protocols were considered as sufficient evidence, macroscopically and microscopically these deductions have been found to be reliable in nearly every case.

CASE I.—202, M. McK., female (12,166). Patient was admitted from Boston, April 27, 1894, aged 73, very deaf and answers questions irrelevantly, wanders about the ward at night, irritable, noisy and extremely

jealous of other patients, filthy in habits, developed a large crop of boils in November, 1898, grew steadily weaker and died December 16, 1898.

Autopsy Report.—Heart flabby, mitral valve thickened, many pleuritic adhesions and right lung showed edema and congestion, capsule of kidney firmly adherent, on section kidney cortex very thin, dura much thickened, pia milky, parietal lobes softened, arterial walls at the base showed numerous yellow patches, all vessels congested, no atrophy.

Assigned Cause of Death.—Interstitial nephritis.

Case II.—409, M. A. R., female (11,078). Patient was admitted from Easton, March 24, 1891, aged 78, third admission, vision much impaired, radials very sclerotic, says she is surrounded by spirits who tease and torment her, very apprehensive, hallucinations of sight and hearing, had appeared to grow gradually weaker for six months and died June 9, 1899.

Autopsy Report.—Body much emaciated, bloody discharge from vagina and rectum, pleuritic adhesions, several hard nodules in upper lobe right lung, heart small, surface arteries calcareous and tortuous, mitral and tricuspid valves calcareous, large gray nodule in liver, multiple nodules in spleen, also in many places in intestinal wall, pancreas contained a hard mass about midway size of hen's egg and firmly attached to intestines and abdominal aorta, capsule of kidney very adherent.

Brain.—Surface of frontal and temporal lobes extremely soft and crumbles, arteries of base marked by calcareous plaques, no atrophy.

Assigned Cause of Death.—Carcinoma of pancreas.

Case III.—860, G. P. P., male (14,537).—Patient was admitted from Duxbury, April 6, 1900, aged 77, very feeble and emaciated, second heart sound very indistinct, pulse too rapid and irregular to count. Has been insane seven or eight years, hallucinations, illusions and delusions. On admission was very filthy and could not answer simplest questions, offered feeble resistance to any care or attention, died April 13, 1900.

Autopsy Report.—Dura strongly adherent to calvarium, when dura was opened four ounces of fluid blood with many clots poured out. The left internal capsule presented an irregular-shaped area of degeneration, grayish-white in color and of jelly-like consistency, vessels of base thickened and dotted with grayish-white spots, no atrophy. Other organs not examined.

Assigned Cause of Death.—Cerebral hemorrhage (sub-dural).

Case IV.—1061, R. B., female (14,711). Patient was admitted September 1, 1900, from Chelsea, aged 92.

Physical Examination.—Almost bald, arcus senilis well marked, skin harsh and leathery, only four teeth remain, vesicular murmurs roughened over all areas of chest, urine normal, vision very poor, tongue tremulous, gait very uncertain.

Mental Examination.—Memory poor for recent and remote events, disoriented as to time, incoherent and rambling in conversation, appetite poor and patient is very feeble, spends a part of each day in bed, grew weaker gradually and died February 23, 1901.

Autopsy Report.—Mitral valve calcareous, also in tricuspid, aorta calcareous, slight passive congestion of lungs, arteries of lungs show atheromatous change, liver atrophic and capsule much thickened, capsule of kidney strips with difficulty having granular surface, few small cysts on surface. Dura thickened but not adherent, vessels at base sclerotic as are all smaller arteries, in first right temporal convolution there is a considerable spot of yellow softening, the tip of the occipital lobe is also softened and slight general atrophy.

Assigned Cause of Death.—Senility.

CASE V.—1116, H. E. R., female (15,248). Patient was admitted November 26, 1901, aged 78.

Physical Examination.—Hair gray and short, arcus senilis present, skin harsh and dry. Heart and lungs not examined owing to lack of co-operation, urine not remarkable, teeth much decayed and scattered, tongue tremulous and deviates to right of median line.

Mental Examination.—Entirely disoriented, says she is 30 years old, cries to go home to her parents, no memory for recent and remote events, quiet but restless, very untidy in dress and personal habits. In February, 1902, suffered from profuse serous diarrhea and died February 17, 1902.

Autopsy Report.—Recent slight adhesions between stomach and liver, many small hemorrhagic spots on mucous membrane of stomach, mucous membrane small, intestine covered with dirty gray mucous, liver cut surface greasy, capsule of kidney strips with difficulty leaving granular surface, many pleuritic adhesions, some thickening of mitral valve, others competent, aorta shows some thickening, edematous softening in parietal and frontal regions, pia strips easily, olfactory tract atrophied, vessels at base show occasional patch of hardening.

Assigned Cause of Death.—Gastro-enteritis and senile dementia.

CASE VI.—1210, G. C., male (15,790). Patient was admitted on January 21, 1903, from Bridgewater, aged 70.

Physical Examination.—Poorly nourished, arcus senilis, subject to dizzy spells, teeth nearly all missing, skin rough and dry, gait unsteady, second aortic sound accentuated, radials sclerotic, lungs shows some moist râles over apex, right; reducible inguinal hernia on left side, urine not remarkable.

Mental Examination.—Much confused, goes about pounding on the doors, disoriented, no insight, memory deficient, general health gradually failing, died May 7, 1903.

Autopsy Report.—Pleuritic adhesions, some excess of fluid in pericardial sac, coronaries sclerotic, aorta shows sclerosis, calcareous deposits and

ulceration; a veil of pus over intestines (Gram-negative bacillus shown); capsule of kidney adherent.

Brain.—Tissue very soft, surface of convolutions roughened and has granular appearance, pia vessels show spots of thickening, vessels at base very sclerotic, slight frontal atrophy.

Assigned Cause of Death.—Peritonitis and senile dementia.

CASE VII.—1350, P. W., male (16,602). Patient was admitted October 18, 1904, from Fall River, aged 70.

Physical Examination.—Very feeble, skin thin and dry, marked arcus senilis, incontinence of urine, breath sounds roughened, loud systolic murmur replacing second sound of heart, arteries much thickened, very deaf.

Mental Examination.—Quiet but restless, clouding of consciousness, blind agitation, disoriented, very restless at night and sleeps very poorly. November 1, 1904, showed rise in temperature, rapid breathing and there were spots of dullness over lungs, died November 3, 1904.

Autopsy Report.—Body well nourished, slight aortic sclerosis, lower lobes of both lungs show hepatized areas with pus points on section, many stones in gall bladder, cyst on surface of right kidney, fatty degeneration in left kidney. Dura strongly adherent externally and internally, pia is milky, middle meningeal artery shows many white patches, area of softening in left hemisphere posterior to postcentral convolution, no other areas of softening, no atrophy.

Assigned Cause of Death.—Broncho-pneumonia.

CASE VIII.—1363, B. B. S., female (15,720). Patient was admitted November 29, 1902, from Taunton, aged 85.

Physical Examination.—Much emaciated, arcus senilis, skin rough and dry, gait feeble and unsteady, murmur heard at apex at beginning of second sound, teeth entirely missing.

Mental Examination.—Quiet, entirely disoriented, untidy, memory equally poor for recent and remote events, very irritable, suffered from multiple abscesses in neck in the summer of 1903 which healed, dysentery in July, 1905, from which patient died July 19, 1905.

Autopsy Report.—Spleen strongly adherent to surrounding tissues, liver shows increase of connective tissue, capsule of kidney strips with difficulty and leaves granular surface, heart enlarged, mitral valves show calcareous patches, aorta calcareous, pusy mucous in the bronchi, dura adherent in frontal region, large vessels at base show calcareous patches and small vessels more uniform thickening, in the first convolutions of right frontal a patch of yellow softening size of dime, no other degenerations, general cerebral atrophy.

Assigned Cause of Death.—Arteriosclerosis and senile dementia.

CASE IX.—1318, M. L., female (15,185). Patient was admitted October 10, 1908, from Fall River, aged 80.

Physical Examination.—Very feeble, hair gray and scanty, arcus senilis,

ankylosis of knees, heart's action very feeble, appetite poor, toothless, skin dry and loose.

Mental Examination.—Quiet, but keeps up a constant muttering in French, disoriented, does not know her age, confined to bed, takes little nourishment, died April 19, 1904.

Autopsy Report.—Mammary glands atrophied, abdominal aorta much sclerosed and calcareous, small cysts on surface of kidneys, pleuritic adhesions, pus points on section of left lung, many pericardial adhesions, tricuspid valve thickened, mitral and aortic valves calcareous.

Brain.—Much softened all over cortex, vessels at base present much atheroma, slight general cerebral atrophy.

Assigned Cause of Death.—Arteriosclerosis and senile dementia.

CASE X.—1561, J. W., female (17,156). Patient was admitted January 10, 1906, from Edgartown, aged 75.

Physical Examination.—Very feeble, skin harsh and dry, prolonged rasping murmur replacing first sound of heart, circulation poor, some arterial hardening, gait feeble, tremor of tongue and extended fingers.

Mental Examination.—Quiet, but restless especially at night, memory defective for recent events, orientation imperfect, delusions of a persecutory nature, irritable, pseudo-reminiscences, suffered from a shock on morning of January 21, 1907, and died January 22, 1907.

Autopsy Report.—Mitral valves much thickened, coronaries sclerosed, lungs negative, strong adhesions throughout abdomen, capsule of kidney strips with difficulty leaving rough surface, numerous cysts just beneath capsule.

Cerebral vessels show marked sclerosis, fresh hemorrhage in right occipital lobe, atrophic patches around the vessels in both hemispheres, slight frontal atrophy.

Assigned Cause of Death.—Cerebral hemorrhage and valvular heart disease.

CASE XI.—1407, T. H. T., male (16,504). Patient was admitted August 8, 1904, from New Bedford.

Physical Examination.—Feeble old man, face asymmetrical, small inguinal hernia, urine normal, lungs negative, soft blowing systolic murmurs heard at apex, arteries somewhat hardened, gait tremulous and unsteady, hands too tremulous to write.

Mental Examination.—Quiet, but restless, constantly arranging his bed clothes, much confused, motor restlessness, disoriented, no change in mental condition, but a gradual physical weakening resulted in death May 7, 1905.

Autopsy Report.—Sacral decubitus, tricuspid valve thickened, also mitral; aorta sclerotic, pleuritic adhesions and streptococcus exudate covering lower lobe left lung, which on section shows much congestion, smears from

the exudate in bronchioles of right lung show pneumococci. Many peritoneal adhesions, slight increase of fibrous tissue in liver, numerous small cysts on surface of both kidneys.

Head.—Grooves for meningeal vessels well marked, dura thickened and adherent externally, brain tissue is fairly soft, a very large cyst in the left choroid plexus; no atrophy.

Assigned Cause of Death.—Lobar pneumonia.

Basal sclerosis was noted in 30 cases and probably that is a small percentage if the microscopic pictures could have been obtained, but that was impossible. In most instances, however, a distinct note was made to the effect that the sclerosis was confined to the large basal vessels and did not penetrate into the brain substance. Four of the 44 brain weights showed distinct loss in weight without any cerebral or basal sclerosis being noted macroscopically, and in only one case of these four did the microscope reveal any thickening of the vessel walls and that was very slight.

Eleven cases showed distinct atheromatous change, no atrophy of the cortex was noted and the brain weights were above the average or only slightly below it.

Six brains macroscopically showed no arteriosclerosis and no atrophy. Of this number one showed a slight thickening of the cortical vessels under the microscope and one distinct cell infiltration around the vessels with the presence of plasma cells, so this case can probably be thrown out altogether.

Conclusions.

I. That the frontal convolutions undergo the most atrophy and that general atrophy is uncommon.

II. That the female brain loses more often in weight and that the loss is greater.

III. That men are attacked by the disease much earlier than women, but live somewhat longer after it is established.

IV. That atrophy does not go hand-in-hand with atheromatous change.

V. That some cases with symptoms pointing to senile dementia show neither arteriosclerosis nor atrophy at autopsy.

IMPRESSIONS OF SOME ASYLUMS IN SCOTLAND.*

By C. A. DREW, M. D.,
Superintendent City Hospital, Worcester, Mass.

Some years ago a member of this Association, who had visited many of the asylums and hospitals for insane in Great Britain and on the Continent, said to the writer: "If you wish to study laboratory methods principally, I advise you to put in all the time you can in Germany, but if I were to be insane I think I would rather take chances in a Scottish Asylum than in any hospital for the insane I know of outside of Scotland and certain small hospitals in our own country." We did not then realize the personal need of asylum treatment but were confident our medical friend had a good eye for the comforts of this world and the thought grew, nourished by reports from other sources, that the asylums of Scotland might be among the most interesting from the clinical viewpoint of all the hospitals for the insane across the seas. So it happened that we found ourselves in the land of "Robbie" Burns for the first time about the middle of September, 1907. The summer had been a rainy one—even for Scotland—as the uncut grass and unharvested grain bore eloquent witness. But September was making amends. Not even the early autumn days of northern New England, nor the late autumn days of our own middle west could have been more delightful than the two weeks in which we were leisurely approaching the Scottish asylums through Westmoreland and Cumberland,—the "lake district of England,"—and the lakes and highlands north of the Forth and Clyde.

We were first introduced to a Scottish asylum at Dumfries, in one of the garden districts of Scotland, a short ride from England's northern boundary. Crichton Royal Asylum is one of the best endowed and most favored of all the Scottish asylums, and for a good first impression one could hardly do better than to visit Dumfries on a fair summer's day. The soft reddish stone buildings sufficiently detached to avoid a crowded appearance, the

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

gently rolling garden and pasture land, like the surface of northern England or our own southern Iowa, with a fertile soil and frequent showers or misty rains, all combined to favor this Royal institution. We were assured that for twenty-five years or more this asylum has had in Dr. Rutherford one of the ablest administrative superintendents of Great Britain.

We did not learn that any effort had been made to make this,—the richest of Scotland's asylums,—a teaching center or a leader in laboratory research work.

In 1907 there were in round numbers 18,000 insane and feeble-minded persons under the General Board of Commissioners in Lunacy for Scotland, about 3000 of whom were boarded out in private dwellings.

The Crichton Asylum is one of the seven Royal asylums all of which, we understand, were financed by legacies, subscriptions and donations prior to the Scottish Lunacy Act of 1857, which marked the beginning of the building of so-called "district asylums" for pauper insane, supported by public taxation.

From 1857 to 1907 Scotland had erected nineteen district asylums, in which, at the latter date, were cared for 9000 of her 18,000 insane. This low average of less than 500 patients per district asylum is in rather marked contrast to the many very large public asylums in England and to a smaller number of very large hospitals for the insane in the United States. It seems to be not unlikely that the smaller asylums in Scotland have made less difficult there the progress in practical psychiatry, so marked during the last two decades.

In addition to the seven Royal asylums, accommodating nearly 4000 patients, and the nineteen district asylums, accommodating upward of 9000 patients, and the 3000 patients boarded out in private dwellings, the report of the Commissioners of Lunacy shows that in 1907 there were 104 patients in private asylums and over 1300 patients in insane wards of poorhouses with restricted or unrestricted licenses.

Scotland has no separate asylum for her criminal insane and had only 51 inmates in the criminal lunatic department of H. M. prison at Perth, January 1, 1907.

The custom in Great Britain is to transfer patients to the Broadmoor Criminal Lunatic Asylum in England and return

them from this asylum to the district asylums in Scotland or the county and borough asylums in England so soon as their sentences have expired. This is in contrast to the practice in Massachusetts, but is in harmony with the practice in New York state, if I am correctly informed.

Partly because the Broadmoor Asylum is a long distance from the prisons and asylums in Scotland, and partly because the insane convict is held in the asylum for the criminal insane only till the expiration of his sentence, it has been found expedient to send to Broadmoor only those convicts serving long sentences and those held "during His Majesty's pleasure," which latter form of commitment gives the patient about the same status as the "court case" has in Massachusetts and New York. As a matter of fact, the influence of English law and the geographical location of institutions, working with other forces, have so eliminated the insane convict from the Broadmoor Criminal Asylum that on January 1, 1907, with a population of 780 (574 men and 206 women) there were less than 50 patients of the convict class in the asylum,—732 patients being held "during His Majesty's pleasure" or the order of the Secretary of State.

On Duke Street in Glasgow are the observation wards for mental cases of the Eastern District Hospital, widely known as "Dr. Carswell's Observation Hospital." This hospital was of peculiar interest because it is the Scottish representative of the much talked of psychopathic hospital of our own land. From the opening of this hospital in June, 1904, with about fifty beds, to May 15, 1907, nearly 1100 patients had been received and treated, with 475 discharged as recovered and 188 discharged as improved. It is interesting to note in this connection that there were 2½ times as many discharged "recovered" as were discharged "improved," and that both combined (663) equalled 61 per cent of the number admitted.

In "Pavalion F" of the Albany (N. Y.) Hospital there were admitted 784 mental cases from February 18, 1902, to February 28, 1906, of whom 211 were discharged recovered and 244 discharged improved. Here we may note that those discharged as "improved" exceeded those discharged as "recovered," in marked contrast to the statistics of the Scotland observation hospital, while the sum of those discharged recovered and those discharged im-

proved (455) equalled 58 per cent of the number admitted. We judge from their respective reports that the types of cases admitted to the Scottish and New York observation hospitals are not very different; alcoholic cases were most numerous in each observation hospital, with a depressive form of alienation a close second as to numbers. Paretics and epileptics were frequently admitted to both hospitals. It seems to us probable that the relatively larger proportion "recovered" from the Scottish hospital and the relatively larger proportion "improved" from the New York hospital represent the respective viewpoints of the individual physicians who pass judgment on the mental condition of those discharged.

The Glasgow Hospital at the time of our visit was as quiet as the wards of any general hospital.

We do not understand that Dr. Easterbrook, Superintendent of the Ayr District Asylum for five years prior to 1908, was the originator of the open-air, rest-in-bed treatment in Scotland. Indeed, we learn from the report of the Commissioners in Lunacy that Dr. Oswald of the Glasgow Royal Asylum was one of the first to introduce this form of treatment there. But to whomsoever the credit of introduction may be due, we found the Superintendent of the Ayr District Asylum a most enthusiastic advocate of this aid to mental medicine. Not only were the acute cases of both sexes resting in bed under a ground-glass covered porch on the south side of a new hospital building for acute cases, but the restless, chronic, noisy and untidy cases were taken from their rooms each morning and put in bed in the open air, sheltered by shacks wide open to the south.

The natural result of removing the noisy and destructive from the building during the day into the open air was to leave the asylum building a restful place for the quiet and infirm cases. The outdoor air of a north latitude tends to make the restless one keep under the clothing, so that the bed-habit is much more readily established, metabolism is enhanced and nutrition improved almost without exception. The high-pitched tones of chronic excitement, so disturbing when confined within echoing walls, hardly disturbs the hyper-sensitive brain when the vibrations are unconfined in the open air. Bath robe and slippers hung on the shack wall and a board walk led to the asylum toilet room. One nurse was having no trouble in attending to the needs of ten or a dozen noisy patients

and we could readily believe that a day in the open air with the increase in nutriment from an outdoor appetite would be a fairly good guarantee of a reasonably quiet night. The method is so simple and the advantages so obvious that we wondered why we had not, from earliest time, kept our noisy and destructive charges in bed through fair weather days in shacks or under an open sky.

There was, it seemed to us, a remarkably effective and pleasing individuality to the new hospital for acute and infirm cases at Ayr. The photographs will give a more comprehensive idea than a pen picture. The hospital is one story and provides 154 beds. "The walls are of white freestone outside and of single brick inside, the stone and brick walling being separated by a three-inch air space. The brickwork is finished internally with Keen's cement below (six feet) and adamant plaster above, all internal angles and corners being rounded off to facilitate cleaning. The floors are in selected narrow maple stained and polished. The floors of bath rooms, conservatories and corridors of the north wings are in terrazzo. The walls of the conservatories are in glazed white brick; the lower walls of the bath rooms and kitchen are tiled and the roofs are slated with red tile ridges."

It would require the full limit of our paper to do justice to this complete hospital, all on the ground floor. You will notice that each ward has its own diet kitchen on the north and sun parlor on the south; that there is a large sun room or "winter garden" adjacent to the reception ward, with a large south veranda which is utilized for the open-air, rest-in-bed treatment, so conspicuous a therapeutic measure at this asylum. Each ward has a few single rooms looking east or west which may be used as isolation or "observation" rooms. Dr. Easterbrook believed with Dr. Clouston that there are periods in the course of certain psychoses of certain individuals when isolation for brief periods is of some benefit to the individual and of much benefit to other patients. In addition to the six single observation rooms belonging to each ward, there is an annex for infectious and contagious cases, at the eastern extremity for women and at the western extremity for men, each complete, with kitchen, bath room, observation rooms, nurses bedroom and a small south veranda. The assistant matron's suite of rooms occupy a convenient central position dividing the women's infirmary wards on the east from the men's infirmary

wards on the west. The assistant matron, it should be remembered, is a graduate of a general hospital training school. She is, in Scotland, a refined woman, responsible to the head matron and superintendent, and paid usually forty pounds a year, less than $18.00 per month of American money.

The infirmary wards are double wards, 84 feet long by 48 feet wide, partially divided by a longitudinal partition ten feet high which is clear of both floor and ceiling for cross ventilation, the upper six feet being of glass and wood to facilitate supervision.

If I were to defend so lengthy a description of this model hospital, I might say that the subject more than deserves the attention given it. Dr. Easterbrook has written,[1] "*The essential principle aimed at in the design of every ward has been to facilitate, structurally, the work of the nurses.*" In our opinion, the aim was high and the result a signal success.

It is about one hour's ride from Ayr to Glasgow and a little more than an hour from Glasgow to Edinburgh. In a sense, Glasgow and Edinburgh are rival cities, beautiful cities both, each almost surrounded by the fertile lowlands between the Firth of Clyde and the Firth of Forth. Both cities have fine buildings, beautiful streets, and noble institutions. Each has its famous university and its well equipped hospitals. If Glasgow may claim preeminence for its population of nearly three quarters of a million and its commerce on all the seas, the gray old capital city may boast of its natural beauty, its history and the larger number of students and scholars it draws from countries of the civilized world. Glasgow has its Royal asylum and large modern asylums of expensive structure for its indigent insane at Gartloch and Woodilee. All these are up-to-date and under excellent management. Edinburgh cares for its indigent insane in a new institution at Bangour, built on the village plan, with cottages more widely separated than the Hospital for Epileptics at Monson, Mass., which is built on much the same plan. This asylum had nearly eight hundred acres of comparatively rough land, more land than any asylum in Scotland except at Dumfries, which latter Royal institution seemed to have the most and best of all material things.

[1] "The New Hospital at Ayr Asylum," Journal of Mental Science, July, 1907.

At Bangour all the cottages were new and the percentage of quiet chronic patients seemed large. About 50 per cent of the patients at this village hospital were sleeping in dormitories without observation at night except that the head attendant slept in the same house. Here earthen "chambers" were provided for the sleeping rooms and there were single isolation rooms with double doors having narrow thick glass panels resembling a large telephone booth, well supplied with fresh air and an electric fan exhausting from the ceiling. The efficient administration of such an asylum through northern winters must, at times, tax the resources even of such an able administrator as Dr. Keay.

A member of our Association approaching the Edinburgh Royal Asylum for the first time must feel a sense of nearing new but familiar things. To some of us whose reading dates back for years "Morningside Asylum" is a more familiar name than any American hospital beyond our own state or personal service. Under the spell of Dr. Clouston's graphic pen, we have known all sorts and conditions of cases at "Morningside." Here has been the leading center for the teaching of clinical psychiatry in Scotland for many years. "Craig House," the new Craig House, a beautiful and costly structure, occupies a commanding view overlooking the city. This, the finest of the Morningside buildings, is most luxuriously furnished in all its parts. Its "Great Hall" for recreation is exquisite in artistic design and may well be the pride of Dr. Clouston's heart.

Morningside has had, and still has, both private and pauper patients. Prior to the opening at Bangour this asylum had been crowded for some years with an excess of pauper patients and the death rate had been comparatively high. Aside from Craig House, Morningside is not so very different from the older private "Retreats" in America.

At the time of our visit to Scotland in 1907, hope was high and expectation keen that in the diphtheroid bacilli discovered by Drs. Ford Robertson and MacRae in the fluids and tissues of persons suffering from general paralysis, a micro-organism had been found which would prove to be the cause of this organic brain disease. Pathologists in England and Germany were mostly skeptical or scornful, it being generally claimed that the bacilli of Robertson and MacRae could be found in the terminal stages of other dis-

eases. It was sometimes admitted that the diphtheroid bacilli might contribute to some of the symptoms of paresis but about the only whole-hearted supporters of Robertson and MacRae were some of our own laboratory workers in the middle west.

Robertson and MacRae were not over-confident. They had experimented long and carefully and were being almost forced to send serum from their laboratory for trial all over Great Britain. They realized that the serum, in many cases, was to be tried by men who had openly disparaged their discovery. Their own investigations were not complete and their findings hardly more than tentative. And yet, certain lower animals, injected with the toxines of the so-called "bacillus paralyticans," developed symptoms as nearly like paresis as could be expected of a rabbit or a guinea pig. A number of cases of far advanced paresis at Morningside had made such remarkable improvement under serum treatment that the Scottish Board of Lunacy and the veteran Dr. Clouston supported Drs. Robertson and MacRae in the strong hope that an antitoxin might be made from these diphtheroid bacilli for the cure of paresis.

This was less than two years ago. Dr. MacRae has since then been appointed to succeed Dr. Easterbrook as superintendent of the Ayr District Asylum. If Dr. Ford Robertson is still pursuing his investigations for the Scottish asylums, we trust he has been provided with better facilities and a more convenient laboratory than he had two years ago.

In marked contrast to the magnificence of Craig House (Morningside) is the Stirling District Asylum at Larbert, where Dr. George M. Robertson had been making a reputation of which any hospital physician might be proud. Among the champions of non-restraint and the advocates of women nurses for insane men, Dr. Robertson is an enthusiastic leader. We went to Scotland with the purpose of making as extended an observation as possible of this institution. The Larbert Asylum is modest in its architecture and much less expensive in construction than the district asylums of Glasgow and Edinburgh. It has only about one hundred and sixty acres of land which would be valued at about $400.00 per acre (forty-eight acres were bought in 1906 for 3900 pounds). Some alienists in Great Britain regard Dr. Robertson as an extremist with a hobby. Some doubt the genuineness

of his non-restraint. Some said his practice would make the male nurse's position humiliating and drive him out of the service.

Retraint is defined by the Board of Lunacy as follows (see 49th Report, p. 16): "Whenever a patient is made to wear an article of dress or is placed in any apparatus which is fastened so as to prevent the patient from putting it off without assistance, and which restricts the movements of the patient or the use of hands or feet, the case should be recorded as one of restraint, irrespective of the reasons which may have led to the use of such restraint or of its having been used in accordance with, or contrary to, the wish of the patient."

Seclusion is defined: "Whenever a patient is placed by day in any room or locality alone, with the door of exit either locked or fastened, or held in such a way as to prevent the egress of the patient, the case should be recorded as one of seclusion, irrespective of the reason which may have led to the use of such seclusion or of its having been used in accordance with, or contrary to, the wish of the patient."

It is, I think, claimed by the officers of the Stirling District Asylum that no case of restraint or seclusion had been used at Larbert during the five years, 1902 to 1906, inclusive. We learn further from the 49th Report of the Commissioners in Lunacy for Scotland (p. 59), that there are three other Scottish asylums in which no case of restraint or seclusion has been recorded for the five years from 1902 to 1906. The three other asylums classed with the Stirling District are the Dundee Royal and Dundee District (under one medical management), the Haddington District Asylum and the Westermains Private Asylum. This report does not show the relative amount of so-called "chemical restraint" used in different asylums, and without a complete knowledge of the amount and frequency of the use of hyoscin and all depressant drugs, comparisons of restraints and seclusions are incomplete and may be misleading. This should be said, however, that our observation at Larbert did not discover the frequent use of depressant drugs. This asylum was more completely dominated by women than any other Protestant hospital for the insane we ever inspected. A head matron had more authority than any resident officer except the superintendent. It was she who engaged and dismissed employees, usually with the approval of the superintend-

ent, for the wards as well as for all the domestic departments. This head matron was a superior woman, a graduate of a general hospital training school who had demonstrated her superiority and been promoted from a ward matron. So generally acknowledged was her fitness that the assistant physicians enjoyed the situation. An assistant physician in another asylum, speaking of the organization at Larbert, remarked ironically, "We would have a sweet time, really, if our matron were given such authority."

In an after-dinner speech at Larbert on the occasion of the dedication of a nurses' home, Dr. Clouston, praising the results at Larbert and claiming credit because Dr. Robertson had been trained at "Morningside," said, in substance, with sly humor: "Dr. Robertson's signal success, to my mind, is due to his keen insight into woman's nature. He who best knows woman's heart obtains best results from woman's service." The laugh was on Dr. Robertson but we are inclined to think there was more than a grain of fact in Dr. Clouston's pleasantry. If such responsibility and authority as were prudently exercised by the head matron at Larbert were given to the wrong woman, there would certainly be a "hot time" if not a "sweet time" in the average asylum.

Through the courtesy of the Stirling District Lunacy Board and the superintendent we were furnished with keys and admitted to all the privileges of an assistant physician at Larbert and cordially invited to stay as long as we wished. We hoped to be useful as a substitute for an assistant during a vacation, but we were delayed past the vacation season, so that the only part we could play of real service to the assistants was that of proxy at chapel service on Sunday. This service was warmly appreciated and accepted as legal tender for all our obligations. We inspected the wards at different times alone as well as with officials. Each building was in charge of an assistant matron. We saw not the slightest evidence of seclusion or mechanical restraint and the hospital atmosphere was very pleasing. The sleeping rooms, when locked from the outside, could all be opened by turning a knob from within so that no patient could be locked in. Only two of the men's wards had men nurses and these were under supervision of an assistant matron.

We had not been accustomed to see patients smoking in bed, but in the infirmary for men at Larbert it was no unusual thing

for the woman nurse to strike and hold a lighted match while the patient in bed would pull at his pipe. When the pipe was working well, she would deftly fit a perforated cap to the pipe to protect the bed and the old man would smile at the white-capped nurse as if she were an angel thinly disguised.

On a certain morning's visit to a men's receiving ward we were interested to watch a muscular young Scot held in bed by four comely young women nurses. He was a mild "manic" case and the expression on his face resembled a grin more than fear or rage. After watching the struggle for a time, we could not escape the impression that his desire to get out of bed might not be so great as his desire to be restrained. Be this as it may, we may safely assume that, when fully recovered, memory of his asylum experience will not be associated with any great dread of another commitment.

In the congregate dining-room, the men and women were not separated, even by screens or banks of flowers, as we have seen in our own country, but were seated at the same table, a man between two women or a woman between two men like guests at a banquet. There was method and a purpose in all this—the purpose being to re-educate and strengthen self-control under natural conditions.

When Dr. Rutherford resigned the superintendency of the Crichton Royal Institution at Dumfries in the summer of 1907, to retire on a pension of 1500 pounds a year, there were half a dozen strong men thoroughly fitted for promotion in the public asylums of Scotland. The chief executives at Ayr, Bangour, Larbert and Perth District Asylums were each and all particularly able, ambitious and progressive men. One was the author of a scientific work on psychiatry of exceptional merit; one had won a wide reputation as a champion of non-restraint and women nurses on men's wards, besides being an able teacher of clinical psychiatry. One had proved his ability by building a large institution on unusual lines and solving difficult administrative problems with signal ability, and one had built an exceptionally good hospital for acute cases and had done much to popularize the open-air, bed treatment of both acute and chronic cases. These were but a few of the props on which honorable reputations had been builded, but to mention these serves our purpose. It was an interesting and

evenly balanced competition. There were also competitors from England. "The remarkable thing about it is," remarked Dr. Clouston, in speaking of the candidates for the superintendency at Dumfries, "that I haven't an idea which one will win, although each one of the candidates was formerly my assistant." It was not till after we reached home that we learned that Dr. Easterbrook had been appointed at Dumfries. Shortly after this came news of Dr. Clouston's resignation and the appointment of Dr. Robertson of Larbert to the superintendency of the famous "Morningside Asylum," Dr. MacRae having been appointed superintendent of the District Asylum at Ayr.

Since the summer of 1907, Great Britain has lost the active services of two strong men: Rutherford, preeminent as an administrator, and Clouston, as a teacher and writer, the peer of any man.

We made no attempt to make an exhaustive study of the asylums of Scotland. Some of them we did not even visit. We saw no evidence that hydrotherapy was much esteemed there, but we feel sure that an adjunct found so useful in Germany and America will not long be neglected. For the spirit of progress prevails in Scotland even as the spirit of liberty was dominant in the days of Wallace and Bruce.

NOTES ON THE TREATMENT OF ACUTE INSANITY.*

By SANGER BROWN, M. D., Chicago, Ill.

So far as its treatment is concerned acute insanity may be defined as a disease which impairs or destroys the patient's capacity to co-operate with, and indeed not infrequently prompts him to vigorously oppose, those who seek to institute measures intended to promote his cure or comfort; it also disqualifies him from conforming to the conventionalities of social, civil or family life so that some special provisions have to be made for his proper care throughout the course of his disorder; practically this usually requires the exercise of such arbitrary authority as can best be applied in an establishment specially equipped for the purpose. In reference to their treatment, patients suffering from acute insanity may be divided into two classes. In the first may be included those in whom there is a morbid excess of bodily and mental activity and in the second those in whom there is a pathologic deficit in these particulars. The former may be boisterous, boastful, blasphemous, obscene, violent and homicidal, the latter apprehensive, silent, hopeless to the point of despair and suicidal. These two characteristics may alternate, if indeed they be not occasionally combined in the same individual. Either excessive or defective activity may be so pronounced, accompanied as they frequently are with persistent refusal of food and insomnia, as to excite serious apprehension of fatal exhaustion.

Acute insanity, aside from that which accompanies general paresis or those cases which from the first show pronounced signs of mental deterioration, tends strongly toward recovery, and the indications for treatment are, therefore, to assist and not retard nature in her restorative efforts.

Treatment in most cases of acute insanity can only properly be carried out in an institution specially adapted to that purpose, and what I have to say is intended to apply to measures therein employed.

* Read at the sixty-fifth annual meeting of the American Medico-Psychological Association, Atlantic City, N. J., June 1-4, 1909.

First, cases presenting excessive activity may be considered. It is always desirable that every patient should be treated individually; for instance if he be noisy he should not be stupefied with drugs because his noise disturbs others; unless, however, special provisions are at hand for his isolation, or perhaps more properly, insulation so far as any noise he may make is concerned, application of the principle of the greatest good to the greatest number may prevail and the individual suffer accordingly. Indeed I may now state that *the main purpose of my remarks is first to call attention to the importance of having such provisions in institutions for the treatment of insanity as will permit the physician to refrain from administering sedative drugs except only when he believes they will benefit the patient to whom they are administered,* with practical suggestions for the accomplishment of this object; and *second, to emphasize the importance of the fresh-air treatment of acute insanity,* with hints as to how this also may be carried out in practice. That fresh air is highly beneficial to persons suffering from tuberculosis has been clearly understood for many generations, and no doubt a few physicians have individually made some special provisions for having their patients kept much in the open air. But it has only been in the last decade or thereabout that a well-defined fresh-air method of treatment has been developed and come to be pretty generally understood and made use of; so, too, doubtless individuals have appreciated the value of the principles herein brought forward in reference to the insane and embodied them in practice.

I think them so important, however, that I should like to see their value generally recognized and methods for their practical application perfected and adopted.

Many cases of the active variety of acute insanity are very noisy and wakeful, especially in the early days or weeks of their attack. Certainly sometimes, if not generally, it happens at the expiration of any definite period, as, say, a week, the patient's physical condition will be far better if sedative drugs are withheld than it will be if the physiological effect of these be maintained. For such patients a room of ample size should be provided, with thick, deadened walls, double doors and windows; this room should be supplied with forced ventilation so as to keep the air strictly fresh even when one or more persons have to remain in

it. In the country where there are no trees or adjacent tall buildings to deflect air currents, the rooms of an institution may be satisfactorily ventilated by the gravity system by using the basement as an air chamber and providing each room with an independent inlet and outlet flue. These rooms, however, which are devoted to the care of noisy patients and, therefore, have to be kept closed, especially in the summer months when a gravity system is inactive, should be supplied with noiseless electric fans in the outlet flue. In such a room, 15 x 12 x 11, so equipped, occupied by two nurses and a patient, the air remains perfectly fresh. I have had such rooms at my disposal now for nearly four years and have come to regard them as almost indispensable in the treatment of certain cases of acute insanity. Incidentally I would suggest that the plan here outlined of supplying an abundance of fresh air is equal or superior to the so-called outdoor treatment in cases of pneumonia, as it involves no increased liability to exposure either of patient or nurse. My experience happily is limited to one case which occurred in a woman of thirty-eight, who made a good recovery while suffering from a very severe attack of acute mania.

The value of keeping a patient in the open air as a means of promoting his physical vigor, sleep, appetite and digestion has been amply demonstrated in the last ten years in the treatment of tuberculosis, and this is of course desirable in all cases of acute insanity, though some are so noisy and unmanageable that it is not practicable to keep them much out of doors. For those patients who are orderly enough to properly permit them to remain out of doors I have found open pavilions facing south, with concrete floors and concrete walks leading to them, well suited for this purpose. They are always dry and, therefore, always available. They may be supplied with comfortable lounges, and in the winter foot-warmers with a fuel cartridge may be used to supply necessary heat. It would, in my opinion, rarely be advisable to attempt to have cases of acute insanity sleep out of doors, as their co-operation could not be counted on to prevent dangerous exposure even if they were not noisy.

While I believe the measures above outlined have a very wide application in the treatment of acute insanity, indeed are of cardinal importance, I do not wish to be understood as intending to maintain that sedatives, hypnotics and hydrotherapy are not useful. In

regard to the latter I wish to say, however, that while some form of it may be employed in many cases with marked benefit, its value by no means justifies the advocacy it has received in some quarters in the last few years as a sort of cure-all, nor the expenditure of huge sums for the installation of elaborate plumbing which has been made in various public institutions throughout the country. Nevertheless, I maintain anyone who studies his cases closely and has at his disposal such provisions as I have described will, I believe, carry some of them at least through to convalescence without the employment of any medicinal sedative, hypnotic or even tonic, or at any rate he is likely to become more and more abstemious in the matter of prescribing medicinal sedatives and hypnotics. A pretty wide experience with both methods has convinced me, as I have said before, that in most cases of acute insanity when insomnia is pronounced the patient's condition is likely to be far more satisfactory, at the end of a stated period, if medicinal hypnotics and sedatives are entirely withheld than if they are freely administered. The disease generally runs a course of several months, and the secretions and consequently the metabolic processes are often profoundly deranged by an attempt to maintain medicinal sedation over so long a period. Indeed it is not difficult to conceive how occasionally such medication might determine a fatal issue or, worse, permanent mental impairment in a case otherwise curable. Finally I wish to remark emphatically that an establishment adapted to the treatment of acute insanity should have rooms measurably impervious to noise and equipped with efficient forced ventilation. It should also be supplied with open pavilions or porches so as to enable certain patients to spend their days wholly in the open air. I believe the medical profession should advocate the installation of accommodations and appliances suitable to the practical application of the principles above described in public institutions devoted to the treatment of acute insanity.

ANATOMICAL FINDINGS IN SENILE DEMENTIA: A DIAGNOSTIC STUDY BEARING ESPECIALLY ON THE GROUP OF CEREBRAL ATROPHIES.*

By E. E. SOUTHARD, M. D.

(From the Laboratory of the Danvers State Hospital, Hathorne, Massachusetts; and the Department of Neuropathology, Harvard Medical School.)

CONTENTS.

* Read in part at the Annual Session of the American Medico-Psychological Association, Atlantic City, N. J., June, 1909.

I have lately possessed the unusual advantage of reviewing a considerable collection of autopsies (247) upon cases of mental disease introduced at various times in the years 1904-1908 at the daily clinics of the Danvers State Hospital, Massachusetts. The cases introduced in these clinics are of particular value, because the often divergent opinions of several diagnosticians, from three to eight or more, are recorded in each instance. The staff has varied from time to time, but has at all times contained members familiar in our recent American psychiatric literature, such as Prof. A. M. Barrett (now of Ann Arbor), Dr. H. A. Cotton (now of Trenton, New Jersey), Dr. H. W. Mitchell (now of the Eastern Maine Hospital for the Insane), Dr. H. M. Swift, and Dr. Charles Ricksher, as well as the moderator, Superintendent Charles W. Page. The clinical diagnoses which I have considered have not been those chosen for the statistical records required for the Board of Insanity, but *all* the diagnoses rendered for each case.

For more minute examination I have naturally resorted to those cases in which all the staff agreed, holding the opinion that uniformity in diagnosis by several men is of more value than that induced by a single chief of clinic.

To establish the degree of accuracy in diagnosis at these clinics, I may mention (what I have dealt with more specifically before the American Neurological Association) the 85 per cent accurate diagnoses where all agreed to general paresis. Only 6 out of 41 cases *unanimously* diagnosed general paresis turned out to be cases of

something else. The reasons for these 6 incorrect diagnoses I shall present *in extenso* elsewhere,[1] but they do not militate against the general accuracy of diagnosis in the Danvers daily clinics.

I was the more astonished to find that, on accepted anatomical criteria, our success in the diagnosis of senile dementia at these clinics was not at all comparable with our surprisingly good results in general paresis. On the hypothesis that cerebral atrophy is a necessary feature in senile dementia, our diagnoses turned out to be only 38 per cent correct. On the hypothesis that cortical arteriosclerosis is a necessary feature in senile dementia, our diagnoses were but 48 per cent correct. And, in fact, 14 of the 42 cases in which the diagnosis of uncomplicated senile dementia was rendered, viz., 33 per cent, proved to show neither sclerosis of terminal arteries nor cerebral atrophy, so that *our general percentage of accuracy, as established by the finding of either cerebral atrophy or cortical arteriosclerosis (or both), was 66 per cent for senile dementia.*

The general analysis of this material may be presented in the following table, which deals with the data of 42 uncomplicated cases in a total of 71 cases of probable senile dementia in a grand total of 247 cases of various types of mental disease subject to anatomical review:

DIAGNOSIS OF SENILE DEMENTIA.

Clinically uncomplicated	42
Brains underweight	29
Males under 1358 G.	10
Females under 1235 G.	19
Brain atrophic	11
At least one-sixth loss in weight.	
Brains atrophic	16
All data considered.	
Sclerosis of cerebral end-arteries	20
Atrophic, not arteriosclerotic	8
Not atrophic, not arteriosclerotic	14

These 42 cases have been subjected to closer analysis with the object of determining the necessary features in a case of senile dementia.

It does not appear that the anatomical and histological characters of senile dementia are as clear-cut and ready of diagnosis

as the post-mortem characters of general paresis. But there is a widely current opinion that the brains of senile dements show well-marked *cerebral atrophy*. Other descriptions give equal prominence to *arteriosclerotic lesions*. Frequently the literary student gains the impression that both kinds of disease characterize the brains of senile dements, or even obtains a thinly-veiled conception that the brain atrophy is somehow due to arteriosclerosis.

Cerebral Atrophy.

With respect to cerebral atrophy, it appears that there is actually no standard by which to determine the amount of loss in weight which shall signify atrophy. Thirteen of the brains in the 42 uncomplicated cases of senile dementia, remarkably enough, yielded weights above the averages ordinarily assigned for male (1358 grams) and female (1235) grams. Thus, unless some of these 13 cases possessed unusually large brains at the outset (of which there is no evidence otherwise), we need consider only 29 cases as belonging possibly in the cerebral atrophy group.

There is, however, no warrant for the diagnosis of cerebral atrophy in every case which is somewhat underweight with respect to the assigned averages of the books, since we must take into account initial differences in brain weight (variations according to race, body weight, stature, age). Thus brain weights ranging from 1461 to 1265 have been assigned to average normal males, and weights from 1341 to 1112 to average normal females.[2]

It is obvious, therefore, that the degree of loss which is to warrant the diagnosis "atrophy" is hard to determine. Perhaps, in the last resort, we should be dissatisfied with any result which did not depend on an obviously impossible calculation—namely, the degree of loss from the original weight in the given case.

If in this series we adopt the arbitrary standard sometimes advocated—namely, a conceived loss of one-sixth the original weight[3]—and take the ordinarily assigned averages as original weights, we at once reduce the cases showing brain atrophy to 11. Thus only 26 per cent of 42 cases diagnosed as senile dementia would show atrophic brains.

Perhaps it is better to trust the qualitative data with respect to the diagnosis, cerebral atrophy. Upon collation of the qualitative findings, paying attention to the occurrence of both diffuse and

focal atrophies of convolutions, the same result as before recorded (on the criterion of weights) was registered for the incidence of brain atrophy. There were 13 cases which could not be regarded as atrophic and 29 which showed either diffuse or focal convolutional atrophy. The distribution of these classes was almost the same as the distribution of the weight classes. Two cases were carried over from the larger class (29) to the smaller (13). (The first on the score of focal atrophy of one frontal region, the second on the score of a focal lesion (possibly aplasia) of the frontal lobes). And two cases were carried in the reverse direction from the smaller class (13) to the larger (29) on the basis of exact gross descriptions, suggesting approximate normality of convolutions.

But the atrophies which were employed to separate these two classes were not all diffuse atrophies such as we have in mind in the current definitions of senile dementia. Only 20 of these cases showed an obvious diffuse cerebral atrophy, and of these four showed a slight or a questionable atrophy. Thus, if the gross qualitative differentiæ are adopted, 16 out of 42 cases of senile dementia (or 38 per cent) showed cerebral atrophy of a convincing character.

Cortical Arteriosclerosis.

A review of the gross and microscopic findings indicates that we cannot safely exclude cerebral arteriosclerosis of greater or less degree in any single case of this series of 42 cases. (Four cases in which no specific note was made of cerebral arteriosclerosis yield other data, gross or microscopic, from which some degree of such change can be assumed.) Similar statements probably hold good of many brains in the non-insane in advanced age.

But it is evident that there are important theoretical differences between cases which show sclerosis of the larger anastomosing arteries and those which show sclerosis of terminal arteries. Sclerosis of the terminal arteries of the brain, whether leading to infarctions or not, obviously means much more from the standpoint of the integrity of the cerebral functions. In fact it would seem difficult to exclude with certainty cases of extensive small-branch arteriosclerosis from the group of organic dementia.

Twenty out of 42 cases diagnosed senile dementia proved to

show a significant arterial disease—sclerosis of terminal cerebral arteries, leading to gross or microscopic infarctions in 16 instances. Although the other 22 of the series showed arteriosclerosis also, this sclerosis was confined to the circle of Willis or to the proximal portions of the primary branches of the circle, so that the changes were deemed of no special psychiatric importance, beyond that which may attach to sclerosis of the pipe arteries throughout the body. Although it may be scarcely justifiable to transfer these 20 cases of small-branch arteriosclerosis bodily over to the group of organic dementia, since many of the symptoms shown may have been due to quite other causes than the arteriosclerosis shown, still it is manifestly unwise to study such cases from the standpoint of a critical symptomatology under the impression that they are pure instances of senile (not organic) dementia.

Cases Without Sclerosis of Cerebral End-Arteries.

An analysis of this residuum of 22 cases not showing significant cortical arteriosclerosis (either focal encephalomalacia or other evidence of sclerosis of terminal cerebral arteries) shows that eight belong frankly in the atrophic series, whereas 14 stand out as cases in which neither diffuse cerebral atrophy nor sclerosis of terminal cerebral arteries can be ascribed to the brains.

Should this prove an adequate analysis of the anatomy of these cases, it is plain that, in the histories of the eight simple atrophic cases, we might find data corresponding to our conception of senile dementia (providing cerebral atrophy is a necessary feature of this disease). And in the histories of the outstanding 14 we might expect a variety of data in which the characteristic features of senile dementia are lacking.

That somewhat similar results might be expected in a large series of cases, at least in so far as the exclusion of arteriosclerotic cases as "organic" is concerned, is seen from the following table compiled from writers, mentioned again below:

Focal Lesions in "Senile Dementia."

Nötzli, 1895	19	13	32
Meyer, 1896	11	13	24
Appeldorn, 1908	18	14	32
Southard, 1910	20	22	42
	68	62	130

Cases of Cerebral Atrophy.

The following pages are devoted to brief clinical summaries of the eight supposedly pure brain atrophy cases, together with the main anatomical diagnoses in each case.

The special features of the different brains are discussed together on a later page.

Case I.—D. I. H., 11801, Path. Lab. 872. Woman, born 1823. *Heredity:* Father and two brothers died of senile dementia. Mother died of asthma. *Personal history*: Normal in girlhood. Taught school. Married 1843; 9 children (four living in 1904); one miscarriage. Of a melancholy turn. Delirious during a fever, 1874. Fractured thigh, 1897, after which came mental change; amnesia, fabrications, apathy. Cut up clothes. Numerous dreams. Fearful, often said she was dying.

Committed to D. I. H., 1904. Emaciation, anemia, senile skin, teeth absent, feet blue, muscles flabby and weak, arcus senilis. Double cataract, blind, pupils non-reactive, somewhat deaf, tongue and face muscles finely tremulous, too weak to walk or stand, slight paraphasia (?), cannot write, radials and brachials thick and tortuous, pulse thready, irregular, and faint (90 to 100), organic reflexes altered.

Disorientation for time, place, and persons. Amnesia for recent and remote events. Impressibility poor. Multiplies as far as 3×5, and adds 2 and 2, but does no further tests. Resistive, restless.

Death 15 days after admission.

The autopsy showed *chronic conditions:*

- Malnutrition.
- Marked general arteriosclerosis, with calcification and ulceration of aorta, extensive coronary sclerosis, and moderate involvement of bases cerebral arteries.
- Bilateral generalized pleural adhesions.
- Scar of right apex.
- Chronic diffuse nephritis.
- Brown atrophy of heart muscle.
- Slight chronic passive congestion of liver.
- Slight chronic localized peritonitis (omental).
- Diffuse cerebral atrophy.

And much evidence of *acute disease:*

- Acute sphenoidal, ethmoidal, and occipital sinusitis.
- Bilateral bronchopneumonia, with acute bronchitis and tracheitis (without lymphnoditis).
- Edema of left side of rima glottidis.

Lung cultures yielded staphylococci, streptococci, pneumococci, and a bacillus of the pseudodiphtheria group.

Culture from the ethmoidal sinus yielded S. p. aureus, but culture from sphenoidal sinus remained sterile.

The *cause of death* was undoubtedly bronchopneumonia.

CASE II.—D. I. H., 11716, Path. Lab. 849. Woman, born 1818. *Heredity* and much of *personal history* unknown. Onset said to be in 1900 after a fall. (Gall-stones and chronic fibrous peritonitis at autopsy.) Widow.

Committed to D. I. H., 1904. Malnutrition, senile skin, arcus senilis, teeth absent, musculature poor, right eye blind, right pupil fails to react to direct light, hearing somewhat defective, left knee-jerk, elbow and forearm reflexes exaggerated, tremor of tongue, labial tremor when talking, right leg weaker than left and kept flexed, incoordination in movements of feet, muscular sense in legs diminished, radials and brachials thickened and tortuous, pulse full, regular, 96.

Excited and resistive on admission. Disorientation for time, place, and persons. General amnesia. Fabricates (assigning wrong names).

The autopsy showed *chronic conditions:*

Obesity.
Aortic sclerosis with ulceration.
Mitral sclerosis.
(Coronary arteries normal.)
Diffuse and focal sclerosis of basal cerebral vessels.
Chronic fibrous endocarditis.
Chronic adhesive pleuritis, left.
Calcified and caseous nodule in central part of left lower lobe.
Scar of right apex.
Chronic nodular perisplenitis.
Cholelithiasis.
Moderate chronic passive congestion of liver.
Fatty liver.
Chronic diffuse nephritis.
Chronic localized peritonitis.
Chronic external adhesive pachymeningitis.
Diffuse cerebral atrophy.

Acute conditions:

Edema of left leg (thrombosis?, not proved).
No cultures were taken from this case and the cause of death is doubtful.

CASE III.—D. I. H., 11881, Path. Lab., 1050. Woman, born 1834. Almshouse transfer to D. I. H., 1904. Nutrition and musculature fair. Senile facies and skin. Arcus senilis. Varicose veins. Teeth almost all absent. Radial arteries not palpably sclerotic. Vision and hearing impaired.

Pleasant, garrulous, Irishwoman, speaking mixture of English and Gælic. Knew month and day of week, but not the year. Repeated Lord's Prayer. Counted from 1-20, performed a few of the simplest problems. Amnesia for remote events, not so marked for recent events. Cannot write or read.

Death in 1906.

The autopsy showed *chronic conditions:*

Cardiac hypertrophy.
Coronary, splenic, and renal arteriosclerosis.

Brown atrophy of heart.
Chronic passive congestion of lungs.
Slight portal cirrhosis of liver.
Chronic diffuse nephritis.
Chronic perisplenitis.
Chronic splenitis.
Acute conditions:
None noted.
No cultures taken.

CASE IV.—D. I. H., 12162, Path. Lab. 1058. Woman, born 1834 (assigned date). Almshouse transfer. Delusions ("holy water taken away"), suspicious, visual hallucinations (talks to supposed faces), violence at night for unknown period before transfer. Committed August 31, 1904.

Malnutrition, teeth absent, edema of feet and lower legs, weakness of muscles, gait unsteady, slight tremor of hands, rather coarse tremor of tongue, vision defective, knee-jerks and Achilles jerks diminished, wrist and triceps brisk.

Disorientation for time (except month). Amnesia complete (could, however, repeat Lord's Prayer). Later complete disorientation.

Death April 24, 1906.

The autopsy showed *chronic conditions:*
Cardiac hypertrophy.
Chronic diffuse nephritis.
Sclerosis with calcification of aortic valve.
Mitral sclerosis.
Aortic sclerosis.
Coronary, basilar, and vertebral arteriosclerosis.
Chronic fibrous endocarditis.
Chronic fibrous pericarditis.
Chronic adhesive pleuritis.
Absence of diploë.
Chronic external adhesive pachymeningitis.
Chronic fibrous leptomeningitis (notably over vermis of cerebellum).
Pedunculated polyp of cervix uteri.
Hypertrophy of right lobe of thyroid gland.
Tumor (probably hypernephroma) of right kidney.
Diffuse cerebral atrophy.
Slight granular ependymitis of accessory sinuses of fourth ventricle.
Acute conditions:
Lobar pneumonia, bronchitis, and fibrinous pleuritis of right upper lobe.
No cultures taken.

CASE V.—D. I. H., 12334, Path. Lab. 1103. Woman, born 1835. *Heredity:* Negative. *Personal history:* Diphtheria and scarlet fever as child. Bright scholar. Married, 1856. Two boys (both alive 1904); one miscarriage. Married again, 1874. Three or four children. Widowed, 1894. Tailoress

till 1903. Corneal thickening of left eye began 1894. Gave up work on account of impaired vision. During 1904 absent-minded, gradually increasing amnesia, visual and auditory hallucinations.

Committed to D. I. H., 1904. Senile skin, slight anemia, teeth poorly preserved, muscular weakness, slight incoordination of finger movements, coarse tremor of tongue, vision absent in left eye and impaired in right, hearing defective. Died October 21, 1906.

The autopsy showed *chronic conditions:*

Emaciation.
Aortic sclerosis with calcification and ulceration in distal half.
Sclerosis of coronary, renal, carotid (expecially distal extremities of internal carotid) arteries.
Mitral sclerosis.
Cicatrices and local ulcerations of ileum (perhaps tuberculous, but not proved).
Chronic adhesive pleuritis.
Chronic diffuse nephritis.
Old scar and local fibrosis of liver.
Cholelithiasis.
Calvarium thin.
Chronic internal hemorrhagic pachymeningitis (right).
Diffuse cerebral atrophy (masked by edema).

Acute conditions:

Lobar pneumonia.
Hematoma of right side of scalp.
Bruise of face.
Cultures from blood and cerebrospinal fluid showed streptococcus pyogenes.

Case VI.—D. I. H., 13442, Path. Lab. 1123. Woman, born 1832. *Heredity:* Father died of tuberculosis at 35. Mother died of old age over 80. One sister became melancholy after loss of four children and died in this condition at 75. Five brothers and two sisters normal, lived to old age. *Personal history:* Normal child. Common school education. Shoe-stitcher of fair capacity. Unmarried. Ceased mill work 1885. After 1885 occasionally nursed for small fees. Gradual physical failure since 1887 (then aet. 55). In September, 1906, worried over selling of relatives' house. Later insomnia, visual and auditory hallucinations, confusion lasting a week. Similar episode in October. Again in January, hallucinations, confusion, delusions of poisoning, disorientation for time, place, and persons.

On commitment, January 4, 1907, senility, vision defective, tremor of tongue, lips, and hands, muscular weakness, slurring and stumbling speech, radial and brachial arteriosclerosis, pulse 120, weak and thready, heart enlarged, organic reflexes defective, amnesia for both recent and remote events, complete disorientation, diminished impressibility. The temperature rose from 99° on entrance to 101° evening of January 8, and to 103° evening of January 14. Death January 15, 1907.

The autopsy showed *chronic conditions:*

Emaciation.
Brown atrophy of heart.
Mitral and aortic valvular sclerosis.
Chronic fibrous endocarditis.
Aortic sclerosis.
Coronary, pulmonary, carotid, and basal cerebral arteriosclerosis.
Small saccular aneurysm of right middle cerebral artery.
Chronic diffuse nephritis.
Chronic gastritis.
Chronic adhesive pleuritis.
Uterine polypi.
Calcified lymph nodes in perirectal tissue.
Hemangioma of liver.
Chronic fibrous pachmeningitis.
Diffuse cerebral atrophy.

Acute conditions:

Bilateral bronchopneumonia with acute bronchitis and lymphnoditis.
Acute splenitis.
Acute typhlitis and colitis.
No cultures were taken.

CASE VII.—D. I. H., 13358, Path. Lab. 1134. Woman, born 1840. *Heredity:* Unknown. *Personal history:* Largely unknown. Attack said to have been gradual in onset; 9 months before admission, about one year before death. Alcoholism probable. Disorientation, vagrant tendency, delusions of suspicion, wandering talk. Emaciation.

On admission feeble, senile skin, arcus senilis, left eye enucleated, cataract of right eye, enlargement of finger joints. Radial arteriosclerosis. Gait tottering. Slight tremor of hands. Brisk knee-jerks and Achilles jerks, plantar reaction slight, abdominal reflexes absent. Delirium and restlessness, with occasional attempts to walk about. Visual hallucinations probable. Was able to obey a few simple directions. Few relevant replies could be obtained. Amnesia for recent events complete; a few isolated facts from past life were recalled.

The patient remained completely disoriented for time, place, and persons; but her attention improved somewhat during the first week after admission. Motor restlessness occasionally appeared during December. Reactions occasionally suggested auditory hallucinations.

Death January 15, 1907.

The autopsy showed *chronic conditions:*

Emaciation.
Carcinoma of pylorus with metastatic nodule in stomach wall.
Brown atrophy of heart muscle.
Chronic fibrous endocarditis.
Aortic and mitral valvular endocarditis with calcification.
Aortic sclerosis with calcification.

Coronary, pulmonary, basal cerebral (including primary branches), splenic, and renal arteriosclerosis.
Slight hydropericardium.
Scars of left apex.
Bilateral chronic adhesive pleuritis.
Chronic splenitis.
Superficial fibrosis of liver, mesentery, and peritoneal wall.
Left eye absent.
Thyroid small.
Irregularity in thickness of calvarium.
Chronic fibrous pachymeningitis.
Diffuse cerebral atrophy.

Acute conditions:

Bronchopneumonia of right lower lobe with lymphnoditis.
Central necroses of liver.
Culture from cerebrospinal fluid yielded *B. coli communis.*

CASE VIII.—D. I. H., 14099, Path. Lab. 1262. Woman, born 1838. According to patient father died at 72 of asthma. Mother at 50 of pleurisy. Two brothers were still-born; one lived 3 weeks. One sister alive at 65. No insanity or alcoholism in the family. At school till 16. Orthopedic operation at Massachusetts General Hospital in 1854. Occupation, housemaid. Since 1906 at Lynn poor-farm. Noisy and troublesome, singing and screaming at night.

On admission emaciation, contractures of legs and right arm, radial sclerosis slight, prominent temporal arteries, pulse small, rapid, of low tension. Sensations adequate with rough tests. Left ankle-jerk and knee-jerk brisk. Plantar reflexes and right ankle-jerks not obtained. Deafness, facial muscles normal. Blind. Incontinence of urine and feces. Stereognosis for many objects good, but patient could not tell a cent from a quarter by touch. Amnesia for both recent and remote events. Memory somewhat better for remote events.

Death August 16, 1908.

The autopsy showed *chronic conditions:*

Emaciation.
Contractures of legs and right arm.
Brown atrophy of heart muscle.
Chronic valvular endocarditis.
Aortic and coronary sclerosis with calcification.
Chronic obliterative pleuritis (right) and adhesive pleuritis (left).
Healed tuberculosis of apices.
Generalized adhesions of peritoneum.
Cholelithiasis.
Chronic diffuse nephritis.
Chronic atrophic hepatitis.
Perforation of right tympanum.
Slight diploë.

Cysts and calcified nodules in both ovaries.
Diffuse cerebral atrophy.
Acute conditions:
Tuberculosis of lungs.
(Tuberculous?) ulceration of transverse colon.
Cystitis.
Sacral decubitus.

General Clinical Analysis.

SEX.

This series of eight cases happens to be composed throughout of women. This fact is in accord with previous experience in the type of senile case termed by Kahlbaum *presbyophrenia*. Systematists like Kraepelin, for example, mention the predominance of females in presbyophrenia, but fail to note any sex differentiation in other mental disorders of the senium (senile depressions, deliria, delusional insanities).

A peculiar fact looking in the same direction was the statistical point made two years ago by Mitchell and the writer,[4] viz., that the female brains accruing from a series of insanities developing in the sixth and seventh decades were more often atrophic (8 of 11) than the male brains (5 of 12). As we then pointed out, the average age at onset of the females (58.4 years) was not much different from the male average age (59.5 years). But the females survived after onset on the average 8.8 years, whereas the males survived on the average only 2.8 years.

Assuming (what has not yet been proved) that a large series of statistics would yield similar results, we should be confronted with the problem how to explain the remarkable viability of females presenting the clinical features of senile dementia and *pari passu* undergoing cerebral atrophy.

A large series of cases is strictly necessary for the solution of this problem. For the moment it is enough to call attention to the somewhat remarkable fact that of all the cases (42) agreed upon as senile dements at the Danvers State Hospital, 1904-8, there were eight cases which showed a relatively pure brain atrophy, and these eight were all females.

Heredity and Antecedent Factors.

The data concerning *heredity* are lacking in four cases. Two cases have no heredity of insanity, whereas two more have some

taint (872, "father and two brothers senile dements"; 1133, "one sister melancholy after loss of children"). Two cases appear to have been born to asthmatic parents (mother of 872, father of 1262).

The history with respect to *antecedent factors* is unobtainable in three almshouse transfers. Alcoholism is probable in one case (1134), the same which showed pyloric cancer at autopsy. The onset in one case is somewhat doubtfully related with a fall four years before death (849), and in one case is more definitely related with a fractured thigh (872). This case of mental disease subsequent to fractured thigh is the case in which we have the best evidence of hereditary taint, in which also we have record of a melancholy disposition throughout life and delirium during fever at the age of 51.

In two cases it appears that there were no upsetting factors (1103, 1133).

It would be of the greatest interest if we knew what relation these conditions bear to the menopause, but the data are lacking.

The important antecedent factors (except heredity) may be briefly enumerated as follows, based in part on clinical history and in part on autopsy findings:

872. Always melancholy, delirious in fever at 51 (old double pleurisy), fracture of thigh at 74, marked general and coronary arteriosclerosis, scar of right apex, chronic Bright's disease, malnutrition, double cataract, deaf.

849. Fall at 82, gall-stones and chronic fibrous peritonitis, obsolete tuberculosis of both lungs, radial, brachial, basal cerebral (no coronary) arteriosclerosis, chronic Bright's disease, obese, blind in one eye, deaf.

1050. Varicose veins; coronary, splenic, and renal arteriosclerosis; chronic Bright's disease; heart hypertrophy; impairment of vision and hearing.

1058. Heart hypertrophy, edema of legs, chronic Bright's disease; aortic, coronary, and basal cerebral arteriosclerosis; thyroid hypertrophy; malignant tumor of right kidney; vision defective.

1103. Corneal thickening at 59, left eye blind and right eye defective at 68, hearing defective, chronic Bright's disease, gall-stones, scars and ulcers of ileum, emaciation.

1133. Physical failure after 55, business worry at 74; radial, brachial, coronary, pulmonary, carotid and basal cerebral arteriosclerosis, chronic gastritis, chronic Bright's disease, organic reflexes defective, vision defective, emaciation.

1134. Alcoholism, pyloric cancer, general arteriosclerosis; left eye enucleated, cataract of right eye; emaciation.

1262. Gall-stones and generalized adhesions of peritoneum, chronic Bright's disease, healed tuberculosis of apices, aortic and coronary arteriosclerosis, blind and deaf.

Social Factors.

The above histories appear at first sight to have little of importance in common. There is, however, certainly little convincing evidence that social factors play much part in the antecedents of these cases (even in Case 1133 the business worry seems to be part and parcel of the disease, as it set in long after the onset of physical failure). Whatever the causes of the brain atrophy, it seems that they cannot be social.

Defects of Vision and Hearing.

But, if social factors are wanting, it is not so certain that individual psychic factors play no part. It is remarkable that all eight subjects showed severe defects of vision, combined in five cases with considerable deafness. The hypothesis lies very near that some part of the mental picture must be related with these defects of the long-distance receptors. Opinions differ as to the capacity of such defects to produce insanity. Congenital or early defects of the sort notoriously fail to produce insanity. But may not acquired blindness and deafness become effective in producing insanity when other senile conditions coexist? The history of case 1103 might suggest this.

On the other hand, it might not be unreasonable to see in the blindness and deafness merely effects of the cerebral atrophy. This is possible, though it must be pointed out that the blindness in some cases was partly peripheral, originating in cataracts in some cases and in corneal thickening in Case 1103. Careful cortical study is essential in the solution of this problem. In any event, we may suspect that a vicious circle exists in these blind and

deaf dements, such that by a process of diaschisis the loss of long-distance perceptions reacts unfavorably upon a mental life already damaged in a variety of ways.

Are there any further common factors in these antecedents? As in the case of heredity and social environment, so too in the case of vicious habits, infectious diseases, and malignant disease, we find no constant or common factors in this small series.

Relation to Manic-Depressive Insanity.

A specific question likely to be raised about any senile mental disorder is, have there been previous attacks? Should we align these cases with the manic-depressive group of Kraepelin? There is, I suppose, no à priori reason why a manic-depressive subject may not eventually succumb to cerebral atrophy. I am disposed to think, however, that the brains of such cases usually stand up well and that manic-depressive cases, when they reach the senium, will be found rather in the arteriosclerotic group or the "normal" senile group than in the group of atrophies now under discussion.

The present small group of cases is in some respects unsuitable for this purpose. 872 is possibly, if by no means probably, a manic-depressive case.

The problem has been approached in an interesting manner by the English Commissioners in Lunacy (1905 and 1907),[4] who have shown that, in 20,680 admissions to English and Welsh insane hospitals (1903), the cases of first and not-first attacks were distributed as follows:

	First Attacks.	Not-First Attacks.
All cases not senile dements.....	13,970	5,546
Senile dements	1,058	106

Making all due allowance for the inadequacy of data in these aged subjects, it would appear certain that senile dementia cannot be made to include a tremendous proportion of manic-depressive cases.

We are reduced, then, in the particular small group of cerebral atrophies here considered, approximately to one of two etiologies—either to inherited tendencies or to causes operating in some concealed fashion in or prior to the senium. Obviously too, these two factors—inheritance and certain senile or presenile agents—may coöperate to produce senile mental disorder.

Arteriosclerosis.

Since our data are thus far neither for nor against inheritance as a factor, we must consider the most prominent senile condition with which we are familiar—arteriosclerosis.

It has been the fashion to ascribe senile dementia, or a great part of its phenomena, to cerebral arteriosclerosis. Perhaps this relations was first asserted most confidently in Forel's clinic, whence Nötzli in 1895 * issued a monograph on senile dementia, without, however, offering microscopic confirmation of the gross relationship asserted. This subject is considered at length in the anatomical sections.

Five of our series showed palpable sclerosis of the radial arteries (one of these, 1262, in which the radial sclerosis was slight showed prominent temporal arteries). Two cases, 872 and 849, were markedly arteriosclerotic clinically, and these proved to show the most marked general arteriosclerosis anatomically.

Three cases gave little clinical evidence of arteriosclerosis, and three more were but moderately sclerotic clinically.

In short, it would be difficult to prove any close clinical relation between arteriosclerosis and senile brain atrophy.

It must be borne in mind, however, that no one of these cases failed to show anatomically a certain amount of arteriosclerosis. In accord with general experience, this sclerosis was of focal and irregular distribution, affecting some organs and sparing others.

The clinical lesson appears to be that neither the presence nor the absence of radial arteriosclerosis offers important clues to the presence of cerebral arteriosclerosis. Not even the presence of severe changes in the radial arteries, or the concomitance of brachial arteriosclerosis, will permit us to divine the degree or even the presence of arteriosclerosis affecting the smaller or even the larger arteries of the encephalon. The same relation holds between peripherally demonstrable arteriosclerosis and, e. g., renal arteriosclerosis.

General Clinical Features.

From the etiological or genetic points of view, we have incidentally considered several general features of these cases, such as the *blindness* constantly present in some degree, coupled frequently

with some *impairment of hearing,* the tendency to peripheral *arteriosclerosis,* etc. These findings may be unified as follows:

Our group consists of eight blind old women, who all presented characteristic signs of senility on inspection, having a senile skin, and, as a rule, also *arcus senilis,* being almost or quite toothless, and diffusely wasted or weak in the voluntary muscular system, and subject as a rule to general emaciation. In short, these subjects could scarcely be distinguished from so many old almshouse inmates, non-insane. Even the blindness would not serve as differentia, as it seems to be grounded in a variety of lesions in the different cases.

Upon further examination, we find no constant situation or degree of peripheral arteriosclerosis, and little agreement in the general characters of the pulse (tendency to increased rate in the more obviously sclerotic cases). The heart is not constantly enlarged, nor are there any constant features upon auscultation. The renal data have not been consistently worked up (partly by reason of difficult collection of urine), but the autopsies have shown always either a chronic diffuse nephritis or renal arteriosclerosis. No other metabolic phenomena have been thoroughly examined. There is certainly no obvious metabolic fault in these cases beyond that which may accompany senility.

Comparatively few signs of focal lesion were made out upon neurological examination:

872. Questionable paraphasia, organic reflexes defective, pupils non-reactive to light (double cataract).

849. Lingual and labial tremor, left knee-jerk, left elbow and forearm reflexes exaggerated, right leg held flexed and weaker than left, incoordination in foot movements, muscle sense in legs diminished.

1050. Not remarkable.

1058. Lingual tremor, tremor of hands, wrist and triceps reflexes brisk, knee-jerks and Achilles jerks diminished, gait unsteady.

1103. Lingual tremor, slight incoordination of fingers.

1133. Slurring and stumbling speech; lingual, labial, and manual tremor; organic reflexes defective.

1134. Slight tremor of hands, abdominal reflexes absent, knee-

jerks and Achilles jerks brisk, plantar reaction slight, gait tottering.

1262. Left knee-jerk and Achilles jerk brisk. Right Achilles jerk and both plantar reflexes absent. Organic reflexes defective. Stereognostic sense impaired (?).

Thus, *tremors, absence* of certain *superficial reflexes, variations* in some *deep reflexes* (tendency to loss of leg reflexes), *defective organic reflexes,* alterations of *gait,* and occasional slight *speech* disorder characterized these patients. Correlations with the details of the protocols and with the microscopic findings has so far not shown adequate reasons for the majority of these clinical findings. 1058 and 1134 seem especially consistent with present neurological knowledge. An examination of the spinal cord in 1134 showed a slight bilateral (approximately equal) pyramidal tract degeneration, as evidenced both by the Marchi preparations and by the neuroglia reaction. This degeneration appears to have been due to the atrophy of the brain which showed " convolutions, except of orbital surface, markedly atrophic, and atrophy especially marked in the Rolandic, parietal, and superior temporal gyri." The Betz cells of both precentral gyri are reduced in number, but the survivors show both axonal reactions and satellitoses. From these findings it may be concluded that such reflexes as those of 1134 do not directly depend upon focal lesions due to arteriosclerosis, but many hang upon atrophic cortical changes, in some instances at least.

Upon the mental side, communication is often rendered difficult through the defective vision and hearing of the patients, and there may be a not unnatural lack of spontaneity in their total relations with the outer world. It is difficult to prove by coarse tests that there is any loss of peripheral sensory power beyond that in sight and hearing; nevertheless such loss or diminution may be suspected to exist.

There is at all events certainly a deficient impressibility or perceptual capacity in these patients, which, coupled with a characteristic incapacity to remember recent events, serves to narrow the mental life to a remarkable degree. Whether the disorientation for time, place, and persons, so frequently noted, is quite constant and inevitable, may be doubted. Perhaps the condition would be

better described as *lack* of orientation, than as positive disorientation.

The amnesia for recent events is constant. Whereas memory for remote events is also frequently deficient, the amnesia for recent events almost always surpasses that for remote events. (One exception, 1050, was an almshouse transfer who had apparently never been able to read or write.)

Both as a consequence of deficient sensory and perceptual capacity and by reason of the marked amnesia for current events, the intellectual life is much reduced in volume as well as devoid of variety.

So far the description qualitatively differs only slightly from what one might expect in the aged, and many writers give the impression that they regard senile dementia as a kind of projection of senility, in which the features of senility will be found to a more pronounced degree.

What is the ground of commitment of these subjects to insane hospitals? In the first place, aside from the question of insanity, these patients are difficult to handle by reason of their muscular weakness, blindness, and deafness, and sometimes by reason of disorder of organic reflexes. Strictly speaking, all these features of the disease could be handled outside of insane hospitals. Possibly 1050 was a case not, in the strict medical sense, insane.

Delusions were made out in four cases (fear of death, "holy water taken away," poisoning, suspicion), but occupy no prominent place among the symptoms.

Hallucinations or *illusions* were demonstrably or probably present in five cases at some stage. One case had before commitment many frightful dreams. The hallucinatory phenomena were as a rule visual or auditory or both, and it is difficult to say whether peripheral or central activity was the more responsible for these findings.

The hallucinations are in part chargeable with bringing about the nightly restlessness and noise of some of the patients, but apparently only in part, since the same restless, noisy, troublesome characters are exhibited by other non-hallucinated patients.

The most constant feature in these patients aside from the exaggerated senile features above enumerated, is *motor excitement.* This was present in all but two cases (1050 and 1103). Restlessness and screaming or howling at night are characteristic. The

patients have spells of motor restlessness, alternating with quiet periods. The violent movements and noisy shouting are apt to come on suddenly and to quiet down gradually. During the spells patients are resistive. The garrulity of old age was exhibited in 1050 only.

AGE, ONSET AND DURATION IN ATROPHIC CASES.

	Age at Onset.	Character of Onset.	Duration.	Age at Death.
872	74	gradual, after thigh fracture.	7	81
849	81	gradual, after a fall?	4	85
1050	70	unknown	2+	72+
1058	61	unknown	2++	63+
1103	69	gradual	2	71
1133	74	gradual	¼	74
1134	66	gradual	1	67
1262	68	unknown	2	70

GROSS ANATOMY OF THE BRAIN.

The pia mater was normal or approximately normal in 5 of 7 cases in which its condition was noted; but in case 1058 it showed a slight diffuse fibrous thickening especially well marked over the vermis of the cerebellum, and case 1262 showed a few small opaque-white spots over the sulcal vessels. There was a well marked compensatory edema of the pia mater in six cases, but in one case, 1103, four days post-mortem, the brain substance had become soft and inelastic and had apparently imbibed most of the pial fluid. The basal cerebral vessels showed sclerosis in five cases; in one of these the sclerosis was confined to the basilar and vertebral arteries. One case showed involvement of the distal extremities of the internal carotid arteries, and in another case only one spot of arteriosclerosis was found in the entire brain, in a secondary branch of the circle of Willis. One case (849) showed a remarkable small varix in the right calcarine region noted and carefully studied by Prof. Barrett.

The brain weights are considered below in a separate table with comparative weights of other viscera. The convolutions, despite the assigned diagnosis of diffuse cerebral atrophy, do not show in all cases a perfectly even wasting. Thus case 872 showed more